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THE RAPID-ROCKING BED: ITS EFFECT ON THE VENTILATION OF POLIOMYELITIS PATIENTS WITH RESPIRATORY PARALYSIS*

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RECENT improvements in the management of respiratory insufficiency in patients with acute poliomyelitis have considerably reduced mortality in this stage of the disease. As a result, increasing numbers of patients have required artificial respiration for long periods after the acute febrile stage of poliomyelitis has passed. Although tank-type "iron-lung" respirators are life-saving for acutely ill patients, their continued use over periods of many weeks or months is associated with certain disadvantages. These include fixation and fibrosis of the diaphragm, restriction of motion of the extremities and difficulty in inducing patients to rely on their own recovering ventilatory mechanisms. A device has been needed that would supply artificial respiration without these disadvantages.

During the past two years, on the Adult Poliomyelitis Service of the New York Hospital, the rapid-rocking bed has been used to supply artificial respiration to many convalescent patients with respiratory paralysis of the spinal type. The bed has also been used in a smaller group of patients with respiratory insufficiency of this type during the acute febrile stage of poliomyelitis. The effect of rocking on the ventilation of the patients in these two groups has been studied.

Introduced by Eve[1] in 1932, rocking has since been shown to be an effective method of artificial respiration through the investigations of several workers.[2-7] Wright[8] reported on the use of rocking for poliomyelitis patients in 1947, but she was largely concerned with the advantages it provided in facilitating physiotherapy and nursing care and made no measurements of ventilation.

The mechanical rocking bed used by Wright was similar to those now proposed for use in poliomyelitis.[9] These beds rock through a smaller total arc than has been used in most of the studies on rocking as an emergency method of artificial respiration. For this reason it was thought that the rocking bed could be used to full advantage for physiotherapy and other care only if its safety and value as a respirator in poliomyelitis were thoroughly studied.

With a shallow tidal volume and rapid respiratory rate, most of an apparently large minute volume would be wasted in respiratory dead-space air exchange. Therefore, in evaluating the effect of rocking on the ventilation of these patients the calculated effective alveolar minute ventilation was used instead of minute volume. This calculation approximated the amount of air actually available to the lungs for gas exchange and eliminated the valueless respiratory dead space, the 120-to-160-cc. volume of the tracheobronchial tree that must be filled with each breath before any inspired air reaches the alveoli.[10-12]

METHODS

Observations were made on 20 patients with spinal paralysis of the respiratory musculature. Five were in the acute stage of poliomyelitis; 15 were convalescing from poliomyelitis but still had respiratory insufficiency. During the time that the observations were recorded each patient studied needed artificial respiration for from four to twenty-four hours daily because of paralysis of the muscles of breathing.

Two rapid-rocking (Respir-aid) beds were used (Fig. 1), equipped with footboards and gatch attachments for the knees and head. They were run at maximum rocking arc, 40 to 44° total (20 to 22° in each direction from the horizontal), except when otherwise stated. The rate of rocking could be adjusted from 6 to 28 complete cycles per minute; rates of from 16 to 24 cycles per minute were generally employed, according to the amount of ventilation produced.

Patients were placed in the supine position on the bed with the knee gatch usually elevated 4 to 5 inches and the head gatch raised no more

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than 2 to 3 inches. Greater elevations of the head or knees restricted diaphragmatic motion and were therefore contraindicated. Placing patients' feet against the footboard and inserting pillows against hips and shoulders prevented sliding on the bed while rocking.

The tank-type respirators employed were standard Drinker and Emerson types, run at pressures and rates indicated. Tidal volume and vital capacity were measured by a Sanborn recording closed-circuit BMR spirometer with mouthpiece and nose clip; the spirometer was calibrated from and periodically checked against a Tissot gasometer.

FIGURE 1. *Patient on a Rapid-Rocking Bed, Showing the Arc of Excursion Employed (Total, 40–44°).*
A = *Bed with foot lowered 20°.* B = *Bed with foot elevated 20°.*

Venous-plasma carbon-dioxide combining power and arterial-blood oxygen saturation were measured by the technic of Van Slyke and Neill.[13] Arterial oxyhemoglobin saturations were followed in some cases by a Millikan ear oximeter[14] standardized against an initial blood-gas analysis (Van Slyke) in each case.

Effective alveolar minute ventilation was calculated in the following manner: observed minute volume minus [respiratory rate × estimated dead space].

RESULTS

Observations during the Convalescent Stage of Poliomyelitis.

Artificial respiration was administered by the rapid-rocking bed to 15 patients who had survived the acute stage of poliomyelitis and were left with extensive paralysis of the intercostal muscles and diaphragm. Numerous measurements of ventilation were carried out on these patients, in the resting state, while rocking and while in tank respirators.

Representative observations on 11 patients reported in Table 1 demonstrate that adequate effective alveolar ventilation was brought about by rocking in every case.

On the basis of ventilation measurements, it was found possible to move poliomyelitis patients safely from respirators to the rocking bed almost as soon as their temperature had returned to normal after the acute stage of the illness. Transfer to the rocking bed was not usually made until patients could breathe without artificial respiration for five minutes. Thus if any difficulty was encountered in transfer the patients could be replaced in their respirators before becoming anoxemic. Those who were most severely paralyzed or who had been most acutely ill experienced some fatigue, anxiety and unsteadiness, which limited their initial periods of rocking to twenty to thirty minutes. After this, however, their adaptation to the bed was as prompt as with other respirator patients, and the period of rocking was increased each day. All patients, no matter how severe their paralysis, enjoyed periods of rocking of at least one to four hours within two weeks of the time they were first removed from tank respirators. Spirometric and oximetric observations repeatedly demonstrated that these patients were receiving sufficient ventilation throughout this period.

In making the actual transfer from respirator to the rocking bed, adaptation to the bed was speeded by coaching the patient to inhale when the feet went down and exhale as the feet came up. It was also found best to speed up the rate of the rocking until the patient experienced subjective satisfaction with the degree of ventilation achieved. Breathing was then measured by spirometer and the rate of the bed adjusted to produce the desired effective alveolar ventilation.

The increase in ventilation brought about by rocking was directly proportional to the magnitude of the arc through which the bed rocked. In a group of four patients (Table 2) the mean tidal volume was increased 45 per cent over resting levels by rocking a total arc of 32°. When a total rocking arc of 40° was used on these same patients, their ventilation was increased an average of 66 per cent over resting levels. For this reason the

largest rocking arc possible (40 to 44° total) was used in treating patients on the rocking bed.

Table 3 presents tidal volumes measured on the same day in patients at rest in the horizontal position, on the rocking bed and in a tank respirator at various pressures. The data given, representative of a large number of similar observations, indicate that the rocking bed at a total arc of 40° did not bring about tidal volumes as great as occurred in the same patient in a respirator at pressures of −12 to −15 cm. of water. For this reason rocking rates faster than the rates usually employed with respirators were often needed to produce adequate effective alveolar minute ventilation.

Figure 2 is a graphic presentation of tidal volumes, observed minute volumes and effective alveolar minute ventilations of patients measured at rest in the horizontal position, while rocking through an arc of 40° and while in a tank respirator at a pressure of −15, +2 cm. of water. The differences between observed minute volumes and calculated effective alveolar minute ventilation are striking and emphasize the unreliability of minute volume alone as a measure of respiratory sufficiency.

Observations during the Acute Stage

The effectiveness of the rapid-rocking bed having been demonstrated in patients recovering from poliomyelitis, an attempt was made to substitute rocking for tank respirators in the treatment of acutely ill patients. Five patients with acute poliomyelitis were carefully selected for treatment with the rocking bed because they had relatively slight or moderate degrees of respiratory insufficiency. Patients with more severe restriction of breathing were still placed immediately in tank respirators.

The effect of the rocking bed on these five acutely ill patients was disappointing. Despite the fact that they rapidly recovered their ability to breathe without artificial respiration *after* the acute stage of poliomyelitis had subsided, *during* the acute stage all but one patient had to be moved from the rocking bed and placed in a tank respirator in order to correct anoxemia, carbon-dioxide accumulation or excessive fatigue. The one member of the group who survived the acute illness on the rocking bed alone experienced severe fatigue and partial disorientation, which would probably have been avoided had she been transferred to a tank respirator.

TABLE 1. *Representative Data on 11 Patients.**

PATIENT	AGE	SEX	DATE	ESTIMATED DEAD SPACE	AT REST					ROCKING THROUGH ARC OF 40°				
					RESPIRATORY RATE	TIDAL VOLUME	VITAL CAPACITY	OBSERVED MINUTE VOLUME	EFFECTIVE ALVEOLAR MINUTE VENTILATION	RESPIRATORY RATE	TIDAL VOLUME	VITAL CAPACITY	OBSERVED MINUTE VOLUME	EFFECTIVE ALVEOLAR MINUTE VENTILATION
	yr.			cc.	/min.	cc.	cc.	liters	liters	/min.	cc.	cc.	liters	liters
O. S.	10	M	9/22/49	100	40	120	150	4.8	0.8	22	200	260	4.4	2.2
			2/ 5/50		28	150	305	4.2	1.4	20	255	405	5.1	3.1
I. P.	18	M	9/20/49	140	44	185	490	8.1	2.0	27	305	700	8.2	4.5
			10/ 4/49		28	185	885	5.2	1.3	22	425	915	9.3	6.3
M. D.	33	M	9/30/49	150	32	230	640	7.4	2.6	22	425	780	9.4	6.1
			2/24/50		22	395	1190	8.7	5.4	20	520	1430	10.4	7.4
G. H.	22	M	11/18/49	140	34	215	245	7.3	2.5	21	400	520	8.4	5.5
			2/ 5/50		27	245	305	6.6	2.8	20	410	655	8.2	5.4
E. S.	24	F	2/ 6/50	140	34	265	670	9.0	4.2	20	425	765	8.5	5.7
			3/17/50		28	215	655	6.0	2.1	16	335	715	5.4	3.1
O. H.	18	M	9/14/50	140	30	170	395	5.1	0.9	16	365	670	5.8	3.6
H. H.	28	M	9/14/50	150	22	275	915	6.0	2.7	16	460	1005	7.4	5.0
M. L.	33	M	9/15/50	140	16	90	185	1.4	0.0	18	335	520	6.0	3.5
E. L.	32	F	9/ 9/50	130	20	360	600	7.2	4.6	16	425	640	6.8	4.7
E. C.	16	M	11/ 2/50	140	30	285	435	8.5	4.3	16	400	580	6.4	4.2
J. F.	20	M	11/ 2/50	150	32	280	490	9.0	4.2	16	410	595	6.6	4.2
Normal values for adults†				120–160	13–20	350–550	3000–5000	4.5–8.0	3.0–6.0					

*Data reported as at room temperature (22°C.).

†These values taken or calculated from the data of Shock and Soley[13] and of Baldwin, Cournand and Richards,[14] corrected to 22°C.

TABLE 2. *Tidal Volumes and Calculated Effective Alveolar Minute Ventilations in 4 Patients.**

PATIENT	AT REST		ROCKING THROUGH 32°		ROCKING THROUGH 40°	
	TIDAL VOLUME	EFFECTIVE ALVEOLAR MINUTE VENTILATION	TIDAL VOLUME	EFFECTIVE ALVEOLAR MINUTE VENTILATION	TIDAL VOLUME	EFFECTIVE ALVEOLAR MINUTE VENTILATION
	cc.	liters	cc.	liters	cc.	liters
G. H.	245	2.8	380	4.8	410	5.4
O. S.	150	1.4	215	2.3	255	3.1
E. S.	265	4.2	365	4.5	425	5.7
H.H.	275	2.7	395	3.9	460	5.0
Mean per cent increase over value at rest			45%	40%	66%	73%

*Data reported as at room temperature (22°C.).

Case Reports

Use of Rocking during the Acute Stage

Case 1. J. L., a 21-year-old woman admitted October 3, 1948, developed progressive paralysis of the extremities, the intercostal muscles and the diaphragm during the first nine days of her illness. On the ninth day she was drowsy and faintly cyanotic even though receiving oxygen nasally. The nauseated, progressively more apprehensive and finally disoriented. After three hours she was transferred to a tank respirator, using a pressure of −15, +3 cm. of water and a rate of 16 cycles per minute, to which she co-ordinated immediately. She remained constantly in the respirator for only forty-eight hours, after which she progressively improved, the respirator being no longer necessary after eleven days. Her vital capacity steadily improved, increasing from 550 cc.

Table 3. *Representative Data on 10 Patients.**

Patient	Date	At Rest			Rocking Through Arc of 40°			Respirator	
		Resp. Rate /min.	Tidal Volume cc.	Vital Capac. cc.	Resp. Rate /min.	Tidal Volume cc.	Resp. Rate /min.	Pressure cm. of water	Tidal Volume cc.
O. S.	9/22/49	40	120	150	22	200	15	−15, +1	305
I. P.	9/20/49	44	185	490	27	305	15	−15, +2	640
								−12, +2	580
								− 5, 0	395
G. H.	11/18/49	34	215	245	21	400	17	−15, +2	580
								−10, +2	520
								− 5, 0	275
E. S.	3/17/50	28	215	655	16	335	16	−15, +2	490
								−10, +2	370
O. H.	9/14/50	30	170	395	16	365	16	−15, +1	580
								−10, +1	515
								− 8, +1	365
H. H.	9/14/50	22	275	915	16	460	16	−15, +1	730
								−10, +1	520
								− 6, 0	245
M. L.	9/15/50	16	90	185	18	335	16	−15, +2	520
								−10, 0	365
								− 5, 0	185
E. L.	9/ 9/50	20	360	600	16	425	16	−12, +2	520
E. C.	11/ 2/50	30	285	435	16	400	16	−15, +2	490
J. F.	11/ 2/50	32	280	490	16	410	16	−15, +2	610

*Data reported as at room temperature (22°C.).

vital capacity was 500 cc., the tidal volume 205 cc. and the respirations 32. She was placed on the rapid-rocking bed at a rate of 20 cycles per minute, which raised her tidal volume to 290 cc. Her cyanosis cleared and she became more alert. Continuous rocking for six days and intermittent rocking for four days more kept the patient free of cyanosis, but she was excessively fatigued and intermittently confused during this period. At the end of this ten-day period she had a vital capacity of 650 cc. and ventilated adequately without artificial respiration. Further recovery was uneventful.

Case 2. P. W., a 19-year-old man admitted August 26, 1949, developed paralysis of the extremities and respiratory musculature during his first two days in the hospital. His respirations rose to 34, and he became dyspneic and apprehensive despite nasal administration of oxygen. He was placed on a rapid-rocking bed to which he synchronized breathing at a rate of 28 cycles per minute. The arterial oxygen saturation (Van Slyke) was 93.3 per cent four hours after he was placed on the bed, at which time he was comfortable and rational. During the next forty-eight hours he developed increasing drowsiness and partial disorientation. The arterial oxygen saturation fell to 89 per cent and the plasma carbon-dioxide combining power rose to 71 vol. from its admission level of 49 vol. per 100 cc. He was transferred to a tank respirator using a pressure of −20, −3 cm. of water and a rate of 18 cycles per minute. His arterial oxygen saturation rose to 95 per cent and his carbon-dioxide combining power fell to 61 vol. He remained in the respirator only sixty hours; at the end of that time he had a vital capacity of 850 cc. Return of function continued to be rapid, and his vital capacity reached 1590 cc. ten days later.

Case 3. J. T., a 14-year-old girl admitted August 30, 1949, had weakness of the extremities, reduction of intercostal muscle activity and only moderate diaphragmatic motion. During the first twenty-four hours after admission she experienced progressive dyspnea. The respirations were 28 to 30 and the tidal volume varied between 90 and 300 cc. She did not appear anoxemic and her plasma carbon-dioxide combining power was 64 vol. per 100 cc. She was given a trial on the rapid-rocking bed. She tolerated this poorly, becoming on the sixth day after being placed in the respirator to 1570 cc. five weeks after the onset of her illness.

Case 4. M. T., a 24-year-old woman, admitted November 15, 1949, developed progressive paralysis of all four extremities, the left side of the face, the intercostal muscles and the

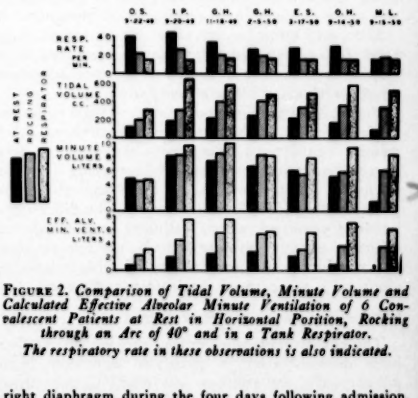

Figure 2. *Comparison of Tidal Volume, Minute Volume and Calculated Effective Alveolar Minute Ventilation of 6 Convalescent Patients at Rest in Horizontal Position, Rocking through an Arc of 40° and in a Tank Respirator.*

The respiratory rate in these observations is also indicated.

right diaphragm during the four days following admission. Her respirations increased to 40. She became drowsy, partially disoriented and exhausted. Her venous-plasma carbon-dioxide combining power, which had been 54 vol. on admission, rose to 73 vol. per 100 cc. She was not cyanotic. She was placed on the rocking bed, which was synchronized to her

respirations at 22 cycles per minute, and immediately became more relaxed and mentally oriented. Arterial oxygen saturation (Van Slyke) after her adjustment to the rocking bed was 98 per cent and the plasma carbon-dioxide combining power 75 vol. By the end of the next twenty-four hours, despite the continued use of the rocking bed and nasal administration of oxygen, she again became irrational and drowsy. The arterial oxygen saturation (Van Slyke) was 95.7 per cent, but the venous-plasma carbon-dioxide combining power had risen to 85 vol. Although ventilation was adequate to achieve oxygenation, it was not sufficient to eliminate carbon dioxide. She was transferred to a tank respirator using a pressure of −15, +3 cm. of water and a rate of 19 cycles per minute. Over the ensuing six hours she became alert, cooperative and well oriented, and the carbon-dioxide combining power fell from 85 to 72 vol. Recovery of respiratory function was rapid. Ten days after being placed in the respirator she was able to ventilate without artificial respiration and had a vital capacity of 680 cc.

Case 5. E. L., a 32-year-old woman admitted August 30, 1950, had weakness of the left arm, the intercostal muscles and the left leaf of the diaphragm. Her vital capacity was 1000 cc. During the three days following admission her vital capacity fell to 500 cc. and her carbon-dioxide combining power, which had been 54 vol. on admission, rose to 65 vol. per 100 cc. She was given oxygen by nasal catheter and placed on a rapid-rocking bed, which was synchronized to her breathing at a rate of 22 cycles per minute. After ten hours of rocking she became fatigued, nauseated and partially disoriented. Her resting vital capacity was still 500 cc. and her carbon-dioxide combining power was 68 vol. The patient was transferred to a tank respirator at a pressure of −15, +3 cm. of water and a rate of 16 cycles per minute. She immediately relaxed and slept, and her carbon-dioxide combining power fell to 60 vol. in twelve hours. For the next four days she required constant artificial respiration, which was furnished alternately by rocking bed and respirator. Thereafter progressively shorter periods in the respirator were needed, and she was able to dispense with all forms of artificial respiration on her eleventh hospital day, at which time her vital capacity had risen to 825 cc.

Comment

During the convalescent stage of poliomyelitis gradually increasing periods of time on the rapid-rocking bed proved to be safe and valuable for patients with respiratory paralysis. The rocking bed aided rehabilitation of respiratory function by providing a bridge between the total ventilatory support of the respirator and the independent unsupported breathing state that is the goal of convalescent treatment for every respirator patient.

The means by which rocking substantially increases ventilation over resting levels is the alternate shifting by gravity of the abdominal viscera; this movement forces the diaphragm up and down in piston-like fashion. A direct relation between tidal volume and the magnitude of the arc through which the bed rocked was shown by the data in Table 2, comparing the ventilation produced by rocking through total arcs of 32 and 40°. Considerably larger tidal volumes were brought about by observers[2-5] who used arcs of 90 to 120°. The difference in results of two recent studies using the Eve method is evidently explained on this basis. Gordon and his co-workers[6, 7] employed a total arc of 40°, whereas Asmussen and Neilsen[5] used 120° and obtained much greater ventilation. An arc of 90° or more seems imperative for use of the Eve method in *emergency* artificial respiration. For poliomyelitis patients, however, a total arc of 40° was the maximum that could be comfortably tolerated when the bed was used daily for periods of several hours.

Advantages of the Rocking Bed

Since it provided excellent ventilation for all convalescent patients, the rocking bed allowed patients early freedom from tank respirators, which they would not otherwise have attained. As patients improved, transient periods on the rocking bed provided all the artificial respiration necessary and considerably shortened the length of time that a respirator was needed. By the removal of patients to rocking beds from the confined space of tank respirators, nursing care, physiotherapy and other therapeutic measures were accomplished more easily. Early full passive joint motion was possible, and contractures of such joints as the shoulders did not occur, as had formerly been the case in respirator patients. Fibrosis and fixation of the diaphragm was prevented, as rocking produced artificial respiration largely by moving the diaphragm up and down. By these various measures it was possible to keep patients' paralyzed musculature in optimal condition in preparation for the possible return of neurologic control of muscle function.

The psychologic aid to every patient was tremendous. Not only did the rocking bed give patients their first feeling of improvement by freeing them from respirators, but it also enabled several patients to conquer their dependence on the respirator and oriented them to greater use of their own muscles of breathing.

Limitations of the Rocking Bed

During the acute stage of poliomyelitis, fever, infection and anxiety increased patients' oxygen consumption and carbon-dioxide production, adding to their ventilatory requirements. The rocking bed, since it brings about less ventilation than tank respirators (Table 3), may be unable to meet the increased ventilatory needs during this phase. Plasma carbon-dioxide combining powers that continued to rise (Cases 2, 4 and 5) after patients were treated with the rocking bed were evidence of underventilation. In addition, the first experience of acutely ill patients on the bed lasted for many hours. As a result they had no opportunity to adjust gradually to the motion, were unable to sleep and sometimes became nauseated while rocking.

For these reasons, during the acute stage of poliomyelitis, patients with respiratory insufficiency due to spinal paralysis were better managed in tank respirators. It was usually thought undesirable to transfer patients from respirator to rocking bed during the acute stage, as this would increase anxiety and might precipitate sudden panic and a

respiratory emergency. Both ventilation studies and clinical experience indicated the wisdom of reserving the rapid-rocking bed for use with convalescent patients.

Value of Spirometry

The repeated determination of tidal volume and vital capacity by spirometer was of invaluable assistance in the management of all patients in the acute and convalescent stages of poliomyelitis. Spirometry, regularly performed on even the most extremely ill patients since 1949, eliminated inaccurate clinical estimations of ventilation and caused no fatigue to the patients. These measurements gave an early and accurate indication of the need for a respirator (adult vital capacity falling below 800 to 1000 cc.) and revealed hypoventilation or hyperventilation in patients actually in respirators. From these measurements respirator pressures and rates could be quickly adjusted to produce physiologically effective minute ventilation. During convalescence repeated tests graphically demonstrated ventilatory improvement and provided an objective basis for determining how soon the respirator could be omitted. When their vital capacity at rest increased to 500 to 600 cc., adult convalescent patients could remain almost an entire day out of a respirator, needing only intermittent periods on the rocking bed. When their vital capacity at rest rose above 800 cc., further artificial respiration was rarely needed.

Importance of Plasma Carbon-Dioxide Combining Power

Repeated determination of the venous-plasma carbon-dioxide combining power gave important information in the evaluation of patients with respiratory insufficiency.[17] Although the need for adequate oxygenation has been emphasized in the management of patients with respiratory insufficiency, the danger of inadequate carbon-dioxide elimination from the lungs has received less attention.

Some of the acutely ill patients who were treated with the rocking bed eliminated carbon dioxide inadequately from the lungs, as demonstrated by rising carbon-dioxide combining powers, even though nasal oxygen administration prevented anoxemia (Case 4). Carbon-dioxide retention with an associated respiratory acidosis[17] may result in confusion,[18] somnolence,[5, 18] hypertension[19, 20] and vascular collapse.[20, 21] These dangers emphasize the need for enough ventilation to eliminate carbon dioxide as well as to prevent anoxemia.[22]

Serial checks of the carbon-dioxide combining power and spirometric recordings will usually indicate whether ventilation is sufficient to expel carbon dioxide fully. When these measurements are not available the reversible narcotic effects of carbon dioxide may be erroneously attributed to "polio-encephalitis," the need for greater ventilation going unrecognized.

Summary and Conclusions

Fifteen patients with respiratory insufficiency recovering from acute anterior poliomyelitis were transferred from tank respirators to rapid-rocking beds as soon as their temperature reached normal and they could breathe unassisted for five minutes. The periods of time spent out of the respirator and on the rocking bed were increased each day. As indicated by tidal volume and vital-capacity measurements, the rapid-rocking bed brought about marked increases in ventilation over that of the resting state. Although rocking produced tidal volumes that were not so great as could be attained by tank respirators, adequate effective alveolar minute ventilations were produced in all convalescent patients.

Substituting the rapid-rocking bed for the respirator early in convalescence facilitated nursing care and physiotherapy and prevented fixation of the diaphragm. Rocking aided the restoration of independent breathing and enabled several patients to overcome their dependence on the respirator.

The rapid-rocking bed was used in 5 patients in the *acute* stage of poliomyelitis with moderate respiratory insufficiency. Four had to be transferred to tank respirators in order to correct fatigue, carbon-dioxide retention or anoxemia. The rapid-rocking bed provided barely adequate ventilation for the fifth patient in the acutely ill group.

Frequent spirometry and serial determinations of carbon-dioxide combining power gave valuable information toward the regulation of artificial respiration in all patients.

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ARTERIAL HYPERTENSION ASSOCIATED WITH ACUTE ANTERIOR POLIOMYELITIS*

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AN ABNORMALLY elevated arterial blood pressure has been a striking finding in over half the patients with acute paralytic poliomyelitis seen at the New York Hospital in the past two years. The hypertension appeared in the acute stage of the disease and sometimes lasted well into convalescence; its degree has in general been proportionate to the severity of the patient's illness.

Although high blood pressure has not been widely commented on as being a concomitant or a sequela of poliomyelitis, the phenomenon has been previously recorded. Grulee and Panos,[1] for example, found transient hypertension in 51 out of 70 children with bulbar poliomyelitis, 18 of 41 children with spinal paralytic poliomyelitis, and 14 of 36 children with nonparalytic poliomyelitis. Lachmund[2] also described hypertension occurring in 22 of 208 patients with poliomyelitis. His patients with hypertension were chiefly severely ill persons with bulbar symptoms; 12 of the 22 died.

Weinstein and Shelokov[3] found prolonged elevations of blood pressure in 7 per cent of 428 patients with acute poliomyelitis. Hypoxia was thought to be the most probable cause of the hypertension in these patients.

Sarnoff and others[4] made the observation that in 4 of their poliomyelitis patients with hypertension and respiratory insufficiency the blood pressure returned to normal levels when electrophrenic respiration was instituted. Others[5-7] who have remarked on the finding of hypertension in acute poliomyelitis have not detailed the distribution of frequency of the phenomenon but have implied that it was associated with severe bulbar disease or respiratory insufficiency.

The observations presented in this report, however, suggest that hypertension is a relatively common associated finding in acute poliomyelitis and may occur in patients with comparatively benign forms of the disease.

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CLINICAL MATERIAL

One hundred and three patients with acute or subacute poliomyelitis were admitted to the adult poliomyelitis service of the New York Hospital during the years 1949 and 1950. Since admission to the poliomyelitis service was largely on the basis of referral from other institutions, there was a high percentage of seriously ill patients, and many had respiratory insufficiency or cranial-nerve paralysis at the time of admission. Ninety-five patients had a sufficient number of blood-pressure readings to be suitable for analysis for this report.

Hypertension was considered present if a patient maintained a diastolic blood pressure of over 90 for a period of more than twelve hours.

Blood-pressure observations were made by the auscultatory method, using an inflatable rubber cuff attached to a mercury sphygmomanometer. Blood pressure was recorded on admission and at intervals of every four hours until forty-eight hours after the end of the febrile stage of poliomyelitis. Any patient whose blood pressure was taken less frequently was excluded from this report. Blood pressures were recorded by members of the particularly experienced nursing staff assigned to the poliomyelitis service. The admission blood pressure and any subsequent elevated pressure readings were checked by a physician. In patients who had developed hypertension, the blood pressure was usually taken at weekly intervals during convalescence until it had returned to normal or until the patient's discharge from the hospital. When possible, follow-up pressures were recorded in the outpatient department after the patient's discharge.

A history of hypertension was sought in each patient. Although many of these adolescents and young adults had recently had school examinations, examinations for military service or examinations for life insurance, only one person (A. W.) gave a history of previous high blood pressure. In this group of patients, of whom none with paralytic poliomyelitis were over thirty-six years of age and of whom 24 were less than sixteen years of age,

the expected incidence of a diastolic blood pressure greater than 90 would be less than 9.1 per cent.[8]

Results

Incidence and Degree of Hypertension

Of the 95 patients, 86 had actual muscle paralysis and 9 were diagnosed as nonparalytic poliomyelitis. The diagnosis of nonparalytic poliomyelitis was made in the presence of an acute febrile illness associated with a stiff neck and back and cerebrospinal-fluid pleocytosis of more than 15 cells. Of the total number of patients, 45 developed arterial hypertension during the acute stage of poliomyelitis. In the entire series, only 1 patient died (of sudden circulatory collapse without respiratory insufficiency) who had not had an elevated arterial blood pressure.

Although the total number of patients was almost equally divided between males and females, severe involvement by poliomyelitis and hypertension developed twice as frequently in males as in females.

Relation of Hypertension to Respiratory Insufficiency and Use of the Respirator

Hypertension was of considerably greater degree and longer duration in patients needing artificial respiration than it was in non-respirator patients. In the hypertensive respirator patients who survived the acute illness the average blood pressure was 164/104 and the average duration of hypertension had already reached ninety-eight days at the time of this report (Fig. 2). In the hypertensive patients not requiring artificial respiration, the average hypertensive blood pressure was 143/96 and lasted an average of five and one-half days. Only 1 patient out of 30 requiring artificial respiration failed to develop arterial hypertension lasting more than

TABLE 1. *Extent of Paralysis and Degree and Duration of Hypertension in 45 Poliomyelitis Patients.*

PATIENT	AGE	SEX	EXTENT OF PARALYSIS	ADMISSION BLOOD PRESSURE	AVERAGE HYPERTENSIVE PRESSURE	DURATION OF HYPERTENSION
O. M.*	36	M	R†, IX, X, XII‡	154/90	180/100	1 day
A. W.*	23	M	R, V, VII, X, XII	160/84	180/100	2 days
J. M.*	34	M	R, VII, IX, X, XII	130/88	180/100	3 days
J. M.	14	M	IX, X	130/86	140/90	1 day
M. D.	34	M	R. A§, A, L¶, L, VII, XII	145/85	160/100	3 months
J. D.*	31	M	R, A, A, L, L, IX, X	230/120	200/120	2 days
J. F.	17	M	R, A, A, L, L, IX	160/90	180/100	3 months**
E. K.*	24	M	R, A, A, L, L, IX, X	110/70	160/90	3 days
J. L.	21	F	R, A, A, V, X	105/65	150/120	3 months**
M. T.	24	F	R, A, A, L, L, VII, IX, X	140/80	170/100	2 months
J. B.	25	F	R, A, A, L, VII	120/80	180/100	6 months**
L. B.*	35	M	R, A, A, L, L, IX, X	140/80	170/110	2 days
J. O.	10	M	R, A, L, X	120/90	130/90	3 days
A. R.	11	M	A, L, IX, XI, XI	130/100	130/100	7 days
S. M.	21	M	A, A, L, VII, IX	127/80	140/100	2 days
J. D.	35	M	A, VII, IX, X	134/92	140/100	5 days
M. T.	15	F	A, A, L, L, IX, X	130/90	140/90	4 days
G. R.	14	M	R, A, A, L, L, IX	150/100	150/100	6 months
E. L.	32	F	R, A, A,	118/84	130/110	8 days
W. N.	29	M	A	132/74	140/90	14 days
A. G.	14	F	A	100/45	140/100	1 day
R. A.	33	M	L, L	130/90	150/100	7 days
F. H.	21	M	L	140/100	140/100	2 days
E. M.	16	F	L, L	142/105	135/95	5 days
S. M.	16	M	L, L	145/100	150/100	21 days
S. B.	16	M	L, L	145/90	160/90	5 days
D. G.*	16	F	R, A, A, L, L	120/80	160/110	1 day
G. H.	23	M	R, A, A, L, L	—	200/120	16 months**
D. J.*	25	F	R, A, A, L, L	136/86	150/105	2 days
R. O.*	19	M	R, A, A, L, L	110/70	170/90	9 days
I. P.	18	M	R, A, A, L, L	150/100	140/100	14 days
O. S.	10	M	R, A, A, L, L	—	180/110	17 months**
J. T.	15	F	R, A, A, L, L	140/90	150/100	8 days
J. W.	17	M	R, A, A, L, L	158/78	170/100	5 days
E. C.	17	M	R, A, A, L	136/100	140/90	4 months**
D. D.	23	F	R, A, A, L, L	130/80	150/100	10 days**
O. H.	20	M	R, A, A, L, L	—	140/100	4 months**
H. H.	29	M	R, A, A, L, L	138/118	180/110	4 months**
J. F.*	17	M	R, A, A, L, L	180/110	235/110	1 day
M. L.	33	M	R, A, A, L, L	—	150/100	13 months**
E. S.	26	F	R, A, A, L	—	170/100	6 months**
S. S.	18	F	A, A, L, L	126/80	150/100	4 days
D. S.	30	M	A, L, L,	118/84	130/100	5 days
B. W.	31	F	A, A, L, L	140/90	140/90	1 day
H. J.	48	F	None	150/95	150/95	3 days

*Died.
†Respiratory failure.
‡Roman numerals = cranial-nerve involvement.
§Arm.
¶Leg.
**Hypertension still present.

Table 1 lists the age and sex, the extent of paralysis, the admission blood pressure, the average hypertensive blood pressure and the duration of hypertension of each of the 45 patients.

The relation of the incidence of hypertension to the distribution of muscular paralysis and the need for artificial respiration are seen in Figure 1. The highest incidence of hypertension occurred in patients suffering bulbospinal paralysis or paralysis of all four extremities. These were the most severely ill patients, many of whom needed artificial respira-

twenty-one days. Of the 10 patients whose blood pressure was still elevated 3 to 12 months after the onset of their illness, all needed artificial respiration for at least two weeks in the early part of their illness.

The part played by anoxemia or high blood carbon-dioxide concentrations in the production of hypertension in these patients is difficult to assess. Similarly, it is difficult to determine whether the respirator per se had an adverse effect on the circulation.

Although frequent plasma carbon-dioxide-combining-power and arterial-oxygen-saturation studies failed to reveal hypercapnia or anoxemia in many of these patients, it is evident that any patient with acute poliomyelitis requiring artificial respiration may suffer at least a brief undetected period of anoxemia or hypercapnia. Such brief periods of anoxemia or hypercapnia may have served to intensify hypertension in these patients. Many patients, however, developed hypertension hours before any significant reduction in their vital capacity, as measured by the spirometer, could be noted.

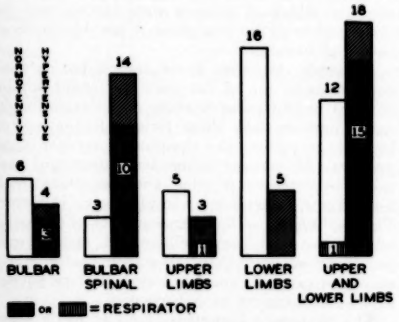

FIGURE 1. *Extent of Muscle Paralysis and Need for Artificial Respiration in 86 Patients with Paralytic Poliomyelitis. In each group normotensives are diagrammed on the left, hypertensives in the right-hand columns.*

Also, brief periods of anoxemia or hypercapnia would not be expected to produce *lasting* hypertension.[9, 10] Anoxemia and hypercapnia appeared to be neither universally present nor the most probable specific causes of hypertension in these respirator patients.

It has been reported that body- or tank-type respirators have a deleterious effect on the circulation.[11] It was difficult to accumulate accurate evidence in the present series that would either support or refute that observation. Artificial respiration was given to all but one of the surviving respirator patients by means of Emerson or Drinker respirators operated at 15 to 20 cm. of water negative pressure and 3 cm. of water positive pressure with each respirator cycle. Artificial respiration was administered to the other patient (J. L.) by the rapid-rocking bed.[12] The patient treated with the rapid-rocking bed developed hypertension as great and as protracted as that of most of the patients treated with tank respirators. However, of 20 patients in whom frequent blood-pressure recordings were made, both before and after the institution of artificial respiration (Table 2), 13 had elevation of blood pressure averaging 34 systolic and 22 diastolic within twenty-four hours after being placed in the respirator.

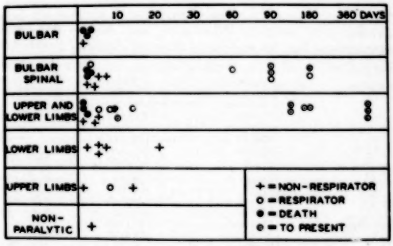

FIGURE 2. *Duration of Hypertension. The symbols represent the number of days that hypertension lasted in each patient. Most respirator patients needed artificial respiration for considerably shorter periods than the duration of their hypertension. "To Present" designates patients who still have hypertension at the time of this report, all of whom required artificial respiration at some time during their illness.*

Three patients with the Guillain–Barré syndrome treated in this hospital in the past year have needed artificial respiration supplied by tank respirators for periods of five days to three weeks. Each of these patients developed hypertension ranging from 160/100 to 230/160 while in the respirator. In contrast to the poliomyelitis respirator patients, whose hypertension persisted for long periods, the blood pressures in the patients with Guillain–Barré syndrome promptly returned to normal when artificial respiration was no longer needed and their neurologic status improved.

Complications of Hypertension in Poliomyelitis

The hypertension in these poliomyelitis patients was a relatively uncomplicated state. Transient papilledema appeared in 3 of the respirator patients with hypertension during convalescence but also was seen in 2 additional nonrespirator patients who did not have elevation of blood pressure.[13] One patient with a blood pressure of 160 systolic, 100 diastolic had a generalized convulsion approxi-

mately one week after coming out of the respirator but had normal kidney function and eye grounds at the time. In addition, he had a family history of idiopathic epilepsy. Blood urea-nitrogen determinations and repeated urinalyses were normal in all the patients with transient hypertension. Of all the patients who have had hypertension for a period of more than three months, only one has an abnormality of blood urea nitrogen, phenol-

TABLE 2. *The Arterial Blood-Pressure of 20 Patients before Institution of Artificial Respiration, and Average Blood Pressure of Same Patients in First 24 Hours of Artificial Respiration.*

PATIENT	BLOOD PRESSURE PRIOR TO RESPIRATOR THERAPY	BLOOD PRESSURE DURING RESPIRATOR THERAPY	CHANGE
M. D.	185/90	180/100	—
J. D.	200/120	200/120	—
J. F.	210/110	235/110	—
D. G.	120/80	160/110	Rise 40/30
D. J.	136/86	170/90	Rise 34/4
E. K.	150/80	160/90	Rise 10/10
J. L.	105/65	140/90	Rise 35/25
O. M.	215/150	150/100	Fall 65/50
R. O.	120/80	174/90	Rise 54/10
L. P.	140/100	140/100	—
O. S.	150/90	180/110	Rise 30/20
M. T.	150/86	170/95	Rise 20/9
P. W.	170/100	140/90	Fall 30/10
J. T.	140/90	150/100	Rise 10/10
L. B.	140/80	170/110	Rise 30/30
J. F.	160/90	210/110	Rise 50/20
D. D.	160/80	230/160	Rise 70/80
H. H.	138/118	190/120	Rise 52/2
E. L.	118/84	130/110	Rise 12/26
O. R.	130/90	110/80	Fall 20/10

sulfonphthalein excretion or urinary dilution and concentration.

COMMENT

Since many of these patients had normal blood pressures on admission to the hospital, and since all who were not in respirators had only transient blood-pressure elevations, it is unlikely that this merely represented a group of hypertensive persons afflicted by poliomyelitis.

Both the hypothalamus and medullary reticular substance contain autonomic nuclei that have vasopressor effects and cause arterial hypertension when stimulated.[14, 15] Involvement of these areas, particularly in the medulla, was demonstrated in several of the patients in this series who came to autopsy. All but 1 of these patients had had hypertension. In the patients who had clinical evidence of bulbospinal or severe upper-spinal-cord involvement, adjacent invasion of these brain-stem autonomic structures could be inferred, and it was in this group that the highest incidence of hypertension occurred.

It was more difficult to relate hypertension to involvement of brain-stem nuclei in patients whose paralysis was confined to the lower extremities. Bodian,[16, 17] however, has demonstrated that damage to brain-stem autonomic structures may occur in any patient with poliomyelitis, regardless of the extent or severity of the clinical signs of the disease. This observation suggests that hypertension may have been the result of central-nervous-system changes even in patients with paralysis confined to the lower extremities.

On the other hand, hypertension as part of a reaction to a situation causing great alarm or apprehension is well documented. It has been produced experimentally under such circumstances[18] and has been seen to occur transiently in association with situations engendering excessive threats to human security such as the Texas City disaster of 1947[19] or in soldiers after long periods of combat duty.[20] Certainly every patient with poliomyelitis is exposed to a situation with terrifying potential consequences. The threat of failure of a resumption of normal patterns of living is present to every patient, and as the disease becomes more severe the patient's very life is endangered. The period of adjustment to a respirator is associated with great fear; it was in such patients that the most marked elevations of blood pressure were seen. Although it was not possible to directly correlate psychologic manifestations of fear and anxiety with elevation of the blood pressure in each patient, the degree of stress to which all patients were exposed was intense and must be considered a possible cause of their high blood pressure.

Anoxemia, excessive blood carbon-dioxide concentrations or use of the respirator could not be indicted as the primary cause of hypertension in many patients, but these factors did appear to intensify or prolong the elevation of arterial blood pressure. No patient in this series developed sustained hypertension who had not been treated with a respirator for respiratory insufficiency. However, Vickers'[21] finding of hypertension in 11 of 44 young adults who had had poliomyelitis many years previously suggests that the non-respirator group may also carry an increased susceptibility to the future development of hypertension.

The temporary hypertension occurring in these poliomyelitis patients may have been due to central-nervous-system inflammation or may have been part of a generalized bodily reaction to prolonged anxiety. The potentially harmful circulatory effects resulting from the use of the respirator or the occurrence of transient anoxemia and hypercapnia appeared to be all that was necessary to convert a transient hypertensive state into a seemingly permanent one. Renal hypertensive mechanisms may have been stimulated in this way to produce an irreversible hypertensive state.

It is of interest in this connection that in 5 of the patients with poliomyelitis and 1 with Guillain-Barré syndrome the blood was examined for the presence of the renal vasoexcitor material (VEM)[2] by Dr. Richard Lee in Dr. Ephraim Shorr's labora-

tory. Significant titers of VEM were found in all patients tested. The titers were exceptionally high in 3 of the poliomyelitis patients and the patient with Guillain–Barré syndrome, each of whom had hypertension and had received artificial respiration. VEM titers were present but lower in the 2 poliomyelitis patients who were not hypertensive. The significance of these observations will be fully apparent only after control tests have been made on other patients with poliomyelitis with and without respiratory distress and without hypertension. Such studies are now in progress and will be published later by Dr. Lee.

Summary

Arterial hypertension, with a diastolic blood pressure greater than 90, appeared in 45 of 95 adolescents and adults with acute anterior poliomyelitis. Males were twice as frequently affected with hypertension as were females.

The highest incidence of hypertension occurred in patients suffering bulbospinal paralysis or paralysis of all four extremities; 29 of the 30 patients requiring artificial respiration developed arterial hypertension.

The blood-pressure elevation in hypertensive patients not requiring artificial respiration averaged 143 systolic, 96 diastolic and lasted a mean of 5.5 days.

The blood-pressure elevation in hypertensive respirator patients surviving the acute stage of the disease averaged 164 systolic, 104 diastolic. The hypertension of these patients has already lasted an average of ninety-eight days, and 10 patients still have hypertension three to twelve months after the onset of their illness.

The appearance of arterial hypertension in these patients may have been the result of invasion of brain-stem autonomic structures by poliomyelitis virus or may have been a manifestation of a generalized alarm reaction. Anoxemia, hypercapnia or artificial respiration appeared to intensify and prolong the otherwise transient hypertensive state that occurred.

The authors wish to express their appreciation to Dr. Harold G. Wolff for critical evaluation of the data and to the nurses of the poliomyelitis service for the many observations they made.

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PENICILLIN ALLERGY*

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PENICILLIN has had wide use since its introduction into clinical medicine, yet severe reactions to this drug have been few although many cases and various types of allergic reactions have been reported. The most common manifestations of sensitivity have been urticaria and angioedema.[1-3] Contact dermatitis among persons handling the drug has also been described,[4-7] and vesicular dermatitis is not rare.[8, 9] Reactions similar to the Arthus phenomenon have come to the attention of two investigators.[10, 11] One group[12] wrote of a tuberculin type of sensitivity, and McClosky and Smith[13] produced anaphylaxis in guinea pigs sensitized to penicillin. Several authors[14-17] have reported anaphylactic shock in human beings due to penicillin sensitivity.

One of the authors of this paper has been the subject of a shock-like type of reaction that followed penicillin therapy. This experience is presented here, as it contains several unusual features and was accompanied by clear evidence of the uncommonly seen atopic type of penicillin sensitivity.

CASE REPORT

A 45-year-old physician who had prescribed and handled penicillin for five years developed numerous furuncles in May, 1949. A colleague prescribed 300,000 units of procaine penicillin given intramuscularly, daily for fourteen days. The organism cultured from one of the furuncles then became resistant to penicillin, whereupon the patient was placed on orally administered aureomycin for twelve days. The furuncles cleared and he remained symptom-free until mid-December, 1949, when multiple furuncles again developed. Treatment with staphylococcus toxoid was instituted, but in late December, 1949, when a new furuncle appeared, the patient decided to give himself 300,000 units of procaine penicillin intramuscularly. Within five minutes of this inoculation a generalized tingling sensation, including pruritus of the scrotum, was noted. The patient observed that his face appeared flushed when he looked in the mirror. At the same time he became unsteady on his feet and began to pant. He also felt a burning sensation throughout his thorax. No audible wheezing was present, however. Within five minutes the reaction subsided and he felt normal again. Three weeks later he noted a small furuncle on his nose. Unmindful of his previous experience, the physician requested a visiting medical student to give him 300,000 units of procaine penicillin G intramuscularly. Within thirty seconds a generalized tingling occurred. He felt hot all over and again noted pruritus of the scrotum. Dyspnea and weakness soon developed, and the patient broke out in a cold sweat. He was forced to lie down. The following observations were made by the physician and the medical student: The skin was at first flushed but then became pale and sweaty. At no time were rales or wheezes audible with a stethoscope. Two minutes after the onset of the shock-like reaction no radial pulse could be felt. Coincidentally, the heart sounds were distant and the blood pressure was recorded as 90/60, falling shortly to a low of 60/40 in contrast to a normal of 120/80. During the following fifteen minutes improvement gradually took place, but weakness continued and the patient had difficulty in walking for several hours thereafter.

Some weeks later, while administering procaine penicillin to a patient, the physician spilled a drop of the material on his finger at the site of a slight abrasion. The area began to itch immediately and soon became erythematous. On the next day he awoke with bilateral conjunctival injection and slight redness and itching of the lower lids. These symptoms disappeared within two hours. Since that time he has taken great care to avoid any exposure to penicillin.

The past history of this patient includes a recurrent duodenal ulcer but no symptoms of hypersensitivity. The family history reveals that the patient's mother and maternal grandmother had severe asthma.

Scratch tests were made in May, 1950, on the volar surface of the forearm, with dramatic results. Penicillin sodium in a strength of 1000 units per ml. and 1 drop of undiluted procaine penicillin G were used for this purpose. Both gave very large positive immediate type reactions. The latter showed a wheal 20 mm. in diameter with one pseudopod 50 mm. in length. These occurred in spite of the fact that the drops of testing solution were wiped off the forearm immediately after the scratch test when the first symptoms of itching began to occur. This preparation of procaine penicillin G consisted of aluminum monostearate and procaine in addition to penicillin G.

In December, 1950, test materials of procaine, penicillin G and aluminum monostearate were prepared, as shown in Table 1. Serial skin tests were then made upon the patient with pure procaine, starting with the highest dilution by the scratch method and terminating with the most concentrated dilution by the intradermal method. All these skin tests were negative. A patch test was next performed with aluminum monostearate. This was also negative at twenty-four and forty-eight hours. These negative tests were interpreted as ruling out aluminum monostearate and procaine as etiologic factors in the therapeutic preparation under consideration. When scratch tests were made with the highest dilution of penicillin G, a small wheal and some erythema appeared. Figure 1 illustrates the size of the whealing reaction and the time interval necessary for its production when the patient was scratch-tested with penicillin G (Solution E). It was not thought wise to perform a scratch test with the more concentrated solutions or to do intradermal tests. Passive transfer tests were then made with the same dilutions of penicillin G and in the same order as had been used for the scratch tests. The normal recipient, who had shown negative intradermal tests to all these dilutions, received 0.10 cc. of the patient's serum intradermally in prepared sites on his arm. On the next day each of these same areas was injected with 0.05 cc. of penicillin G (Solution E). A wheal and an area of erythema appeared within 10 minutes, demonstrating the production of a positive passive transfer test to penicillin G in one person. Passive transfers on two other subjects were negative.

DISCUSSION

From the history and physical examination, it appears that this patient suffered a shock-like reaction occurring immediately after an intramuscular injection of penicillin G procaine. Scratch and intradermal tests for procaine were negative; scratch

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and patch tests with aluminum monostearate were also negative. However, scratch tests with penicillin G were positive. We therefore have strong evidence that penicillin was the causative factor. Since the skin reaction to penicillin was immediate and in view of the fact that the reaction itself began immediately after the injection of the penicillin, we have justification for regarding this reaction to penicillin as belonging in the immediate wheal-reacting type of allergy. The positive passive transfer (Prausnitz-Küstner) reaction lends support to the likelihood of circulating antibodies to penicillin in the patient's serum.

The patient's symptoms and signs suggested a form of circulatory shock. Several theories have been advanced to explain this phenomenon. Among them, Knisely et al[18] hypothesized a separation of several sets of pathologic mechanisms that may act during circulatory shock. Gurd and Roberts[19] reported the autopsy findings in a patient who died of shock after an injection of antitetanic serum. The vessels of the splanchnic area and the right ventricle in this case were distended and filled with blood. The liver was congested, as were the spleen, pancreas and kidneys. Sheppe[20] reported a death after tetanus antitoxin administration; autopsy showed similar engorgement of veins of the head and neck, a right heart full of blood and a liver engorged with blood. These cases might well fall into Knisely's reservoir-retention-shock category. In other words, blood may be stored in some organs by action of spasm of certain vessels. In the case under discussion this spasm may have been only a temporary one.

Modern concepts of allergy indicate that histamine or a histamine-like substance is released when antigen and antibody combine. This has not been conclusively proved, however, up to the present time. If histamine is released, it is possible that the mode of action is concerned with causing certain vessels to contract so that blood is stored in various organs in sufficient quantity to bring about shock-like symptoms similar to those observed in the present case.

Whatever the mechanism of these acute shock-like allergic reactions may be, their clinical occurrence warrants emphasis on the following points: Penicillin is widely used and is a frequent sensitizer; increasing experience with newer antibiotics confirms their sensitizing potentialities. Such observations in themselves should cause the physician to weigh carefully the necessity for the employment of these drugs in each case. The prescription of an antibiotic for a mild affliction when it is not clearly indicated on chemotherapeutic grounds may deprive the patient of the benefit of the same drug at a later period when indications for its use are obvious.

Hypersensitivity, when it does occur, is usually of the delayed type, with symptoms appearing after a period of days or weeks. These symptoms may be mild or severe but can eventually be controlled by stopping the drug and by administration of appropriate antihistaminic and topical therapy.

TABLE 1. *Solutions Used in Skin Tests.*

SOLUTION	SUBSTANCE	AMOUNT OF SUBSTANCE IN PHYSIOLOGIC SALINE
A	Procaine	3 gm. in 5 cc.
B	Solution A	1 cc. Solution A in 9 cc.
C	Solution B	1 cc. Solution B in 9 cc.
D	Penicillin G	200,000 units in 5 cc.
E	Solution D	1 cc. Solution D in 9 cc.
F	Solution E	1 cc. Solution E in 9 cc.
Patch Test I	Aluminum monostearate	A pinch, moistened with buffered phosphate solution

FIGURE 1.

The possibility of the appearance of an immediate or atopic type of allergic reaction must never be overlooked. Here detailed questioning in regard to an allergic background and previous experiences with similar preparations may avoid not only prolonged discomfort for the patient but a serious and possibly fatal accident as well.

Summary

A case of the atopic type of penicillin sensitivity is presented in a patient who appeared to suffer circulatory shock.

Scratch tests were strikingly positive to penicillin G.

A successful passive transfer test was obtained, indicating the presence of circulating antibodies to penicillin.

One possible mechanism of circulatory shock is discussed and the potential dangers of this type of reaction emphasized.

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THE PHYSICIAN'S SOCIAL RESPONSIBILITY*

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BOSTON

EARLY in the second hundred years of our society's existence, it may be fitting to inquire into the purposes that bind us into an organization that has existed over a century, — and, through our parent society, considerably longer.

If we examine the earlier clauses of the by-laws of the Massachusetts Medical Society, we see clearly that the original purpose was scientific, — to hold discussions and disseminate information about the management of cases that physicians were encountering in their practice. This continues to be one of our important activities, not only at the local district level, but also in the state and national societies. However, through the years many problems of diverse nature have arisen that affect physicians as a group and require discussion and action.

I have chosen as my subject "The Physician's Social Responsibility." This means his responsibility both to his fellows and to his patients and the public, for it is amply demonstrated that what one member does for good or ill brings credit or discredit on all of us.

During the last few years we have found our profession, particularly organized medicine, on the defensive, the object of a barrage of criticism from many directions. The reasons and motives behind this wave of criticism are not altogether clear, but I have little doubt that the sins of a few in such matters as fees have brought all into disfavor. It has become a popular sport for many authors, both lay and professional, to take a fling at us for all sorts of sins of commission and omission. To a certain extent this is to be expected, since anything to do with health has become very important on the priority list of items of news value, and in a democracy like ours any group that is important to the public welfare must expect that the right of free speech will be fully expressed in its direction. If there is any doubt in anyone's mind that the medical profession as a whole is important to the public welfare, let us simply point out that for the first time in history a professional group has been singled out for special congressional legislation in connection with Selective Service and that all physicians up to fifty years of age are now registered with the Federal government. Has there been any similar treatment of lawyers, ministers, engineers or teachers? In all the preparation of civil defense

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it is again the medical profession that is singled out for special action, and we may soon expect that each of us will be ticketed with a special job to perform in the event of emergency. Authority exists to commandeer our services and to keep us under that authority as long as the emergency exists. Is this required of any other groups except police and fire departments, who are already under the civil authority? Yes, we must recognize that, although we operate as individuals and remain one of the most ruggedly individualistic groups in the country, we are public servants because we have something special and vital to contribute, which others cannot. It is fitting, therefore, that we should look into our organization to see what reasons may exist for all this clamor against us.

Through the years we have formulated a "Code of Ethics," which has been modified from time to time, and which forms the basis for our conduct toward each other. During the past two years I have served with the committee of the State society whose function is to interpret that code in relation to complaints that have been brought against our members. Its deliberations are of course secret, but I believe I can, without violating any confidences, outline some of the problems that have come before the committee and point to some of the pitfalls to be avoided if we are to stay out of trouble.

In recent months the scope of the committee has been somewhat enlarged through the urging of the American Medical Association that the existence of a grievance committee be publicized, so that the public may know where to bring any complaints against physicians. After this was published in the newspapers there was a sudden flood of complaints of all sorts, and since then there has continued a steady flow of communications expressing all sorts of grievances.

It is of some interest, and perhaps to be expected, that a considerable proportion of the initial flood of complaints came from inmates of institutions for the care of neuropsychiatric patients. There may have been grounds for some complaints, but in most instances they obviously represented part of the symptomatology of the writers' psychoses. Another considerable group came from persons who appeared to be equally psychopathic but were not confined in institutions. We have all met such patients in our practice. They are the sort who shop around from one physician to another, never obtaining any satisfaction, usually paying no bill and having no insight into the neuropsychiatric nature of their somatic complaints. They are misfits, who seem to derive a certain pleasure out of puzzling various doctors with intractable symptoms.

However, others had legitimate complaints of varying degrees of gravity. Many were not really questions of ethics but often represented misunderstandings or failure to have any agreement on fees before a considerable obligation was incurred. Percentage-wise there have been more complaints about charges than about any other item. The committee has tried, as far as possible, to get the parties to settle their own differences, and in most cases this has worked out satisfactorily. In many instances the trouble has arisen because a patient, on his own initiative, has gone directly to a specialist without consulting his family physician about the necessity for any such specialist. The specialist, not knowing the patient's financial status, has often given expensive service when the patient should really have gone to a public clinic. In a few instances there has been what the committee considered outright overcharging, and in such instances the committee has not hesitated to tell the doctor so. Considering the number of doctors' bills that are sent every month, I think there have been singularly few complaints.

In several instances there have been complaints of physicians' demanding cash payment on the spot when answering emergency calls. The committee has no objection to the doctor's asking for his fee under such circumstances, but in some instances the way in which it was done gave the family the impression that the doctor was more concerned with collecting his fee than with looking after the patient's welfare.

We have had complaints from druggists that physicians have made unpleasant remarks about their service, or that there had been an error in filling a prescription. In one instance it was definitely established that the physician had made the error, and an apology was demanded by the committee.

More recently there have been a number of direct complaints about a doctor's professional work — careless examination, wrong diagnoses and so forth.

Our most difficult cases have had to do with strife within hospital staffs, and have confronted the committee with the problem of supervision of physicians' work or limitations on the type of work that a physician is permitted to do within a hospital.

This subject, which comes under the over-all heading of a "medical audit," has grown into a very important one in which public interest is very much concerned. Who shall decide whether a physician is qualified to do a certain type of work, — especially in surgery? The State grants licenses to practice medicine but does not limit in any way the type of work that a licensed physician shall do. Of course the public has the right to sue for malpractice when treatment has not been according to generally accepted standards, and this is in a sense a check on physicians' attempting treatments for which they are not qualified, but this is not an altogether satisfactory way of dealing with this situation.

We are all familiar with the setup of the specialty boards, and we have heard all sorts of opinions

about their requirements and their mode of operation.

In most hospitals it is the recognized responsibility of the trustees to grant privileges within their hospitals, but they must have some method of being advised about who is capable of doing what. In house and office practice the problem does not arise, but when a patient is hospitalized another party, — the hospital board of trustees, — has a responsibility for the patient's treatment, even though he may be a private patient. By various court decisions it has been established that trustees have authority to appoint staff members according to their own established policies, and that these appointments may be terminated at any time at their pleasure. In malpractice suits by private patients, hospital trustees have been made joint defendants because they granted the doctor the privilege of treating the patient in the hospital. By the same courts it has also been held that staff committees who made the recommendations to the trustees are equally responsible. Therefore it is a serious responsibility to sit on the staff committee that recommends appointments to the trustees, and members must exercise independent judgment on the qualifications of various physicians, without being influenced by considerations of friendship or fear of reprisals.

Different hospitals have somewhat different methods of representation on their executive committees. To a certain extent they are mostly based on the models set forth by the American College of Surgeons, which has been handling the matter of hospital standardization and certification for a number of years. In some instances the majority of the committee are chiefs of departments appointed more or less permanently by the trustees; in others the majority are elected by the staff at large for a short term of years. In almost every case some members of the committee are elected by popular vote, and this may result in a certain amount of staff politics. In the hospital that I represent appointments are made on a broad democratic base, with the majority elected by the staff in open meeting, yet we have never had any serious controversies, and the staff has usually made its selections wisely.

The initial granting of staff membership and the definition of privileges usually present no great problem, for nearly all applicants nowadays are exceedingly well trained in their chosen field, hence there is usually no question about their qualifications. Board certification is always given consideration but is by no means an absolute requirement for privileges, even in a special field. The renewal of privileges granted long ago to some of the older staff members, whose original training was much less extensive, is a more serious problem. Under current standards these men probably could not obtain admission if they were new applicants, but the withholding of privileges previously granted is a matter in which legal action for redress could be taken.

It is nearly always about surgery that controversies arise, including both the privilege of performing operations and that of assisting surgeons. This appears to be principally because the public has been willing to pay larger fees for surgical procedures than for the diagnosis and treatment of medical conditions, even though these may sometimes be quite complicated and may call for quite as much special training as surgery. The temptation to perform operations for which one is not really qualified is a very strong one and is one of the bases for the current movement to have physicians work on a salary basis rather than on the traditional fee-for-service basis.

In many parts of the country a strong movement has arisen, — partly the result of hospital policies about board certification, — to have "general practice sections" set up in hospitals. There has, however, been no agreement so far on what constitutes "general practice" and what general practitioners should be allowed to do, particularly in the field of general surgery. In obstetrics it is relatively easy to define the situations that call for the services of a specialist trained in obstetrics, but general surgery is such a broad field that rules are difficult to establish. Even in the matter of assisting a surgeon in the operation or in after-care, it is not easy. In our hospital it is a principle to grant privileges of assisting in surgical operations only to those with sufficient training to carry on the operation if anything should happen to the surgeon. In aftercare, too, there are plenty of problems concerning such things as fluid and electrolyte balance, with which many general physicians are not well equipped to deal effectively.

Nor are these problems confined to surgery. Should the handling of diabetic acidosis or of acute cardiac failure, especially associated with a complex type of arrhythmia, in which a choice between various potent drugs is involved, be left, entirely unsupervised, in the hands of the general physician? These are complex questions that concern, in their final analysis, the quality of the conscience of the physician and his ability and willingness to appraise his own limitations. Often the decision of the staff committee must be based not only on the training that a candidate has had but on whether he is considered a "safe" man. A general physician once remarked that whenever he was called to see a patient he asked himself three questions: Is it serious? Can I handle it myself? Does it need to be hospitalized? Such thought processes make him a safe man, from our point of view.

There are various ways in which a man's work can be checked, several of which have been in use for years. One is the requirement that all tissue removed at operation be submitted to the patholo-

gist for examination. Another is the custom of having all deaths and complications reported and discussed at monthly staff meetings. From some quarters there has been pressure for a more formal and intensive medical audit, in which all records would be carefully scrutinized to see whether the diagnosis made was justified by the evidence given and whether the treatment ordered was appropriate. In military hospitals, and in those clinics directed by one man, this would be comparatively simple, but in the ordinary community hospital it would involve great difficulties. The fact remains, however, that concerning hospitalized patients, who presumably have some serious difficulty, the public has a right to expect some degree of supervision of every physician's work, in order to secure their safety.

The physician, too, has his problems of responsibility for matters not completely under his control. Can he be sure that the blood bank's blood typing will always be correct, that other laboratory determinations are correctly done, that the sterile supplies that he uses are properly prepared, that the orders that he gives to nurses for the administration of drugs are properly carried out? In lawsuits the physician himself is held responsible for all these things, many of which are administrative matters.

The operation of a modern hospital has therefore become a very complex affair, in which there must be harmonious and effective co-operation between trustees, administrator and staff and in which no one party can expect to dominate the situation. Most of the serious controversies, — and many have become so acrimonious that accusations of unethical conduct have been brought before the committee, — have arisen because of the failure of the various parties concerned to meet regularly to discuss their common problems. Sometimes a dominating superintendent has been at fault; sometimes it has been a faction of the staff that has persuaded the trustees to dismiss the members of another faction. One of the best recent developments in our hospital was the establishment of regularly scheduled meetings between representatives of the trustees and staff, with the director present. Many problems have been solved at these joint meetings.

What has all this to do with the district medical society? It has much to do with it, for that is the medium through which doctors as a group can protect their interests by formulating a common policy. If any doctor, or group of doctors, has reason to believe that they have been unjustly used by a hospital administrator or board of trustees, they can report the facts to the district society, which can take action either by direct discussion with the parties concerned or by referring it to the State society. If no solution is reached by these means, the problem can be taken to the Judicial Council of the America Medical Association, which can advise the Committee on Education and Hospitals that the hospital concerned should be removed from the list of hospitals approved for intern and resident training. This is a powerful weapon that can be held over the heads of intransigent hospitals.

Finances again are at the root of many controversies within hospital staffs. Hospitals running at a deficit see opportunities to make a profit by putting certain types of staff members on a salary basis. The status of pathologists, anesthesiologists and radiologists is still not clearly defined. There is something to be said on the side of the hospitals, because in these departments the hospital grants a virtual monopoly on all this work to one person and often provides much expensive equipment. There is also something to be said for the hospital that is running at a loss because of providing expensive equipment and personnel to create an ideal workshop for physicians who derive handsome incomes therefrom. If either private or government funds do not come to the assistance of voluntary hospitals it may become necessary to ask the physicians to carry some part of this burden. Certainly if the hospitals went under the physicians, as well as the public, would suffer. One can readily appreciate that the public might think that there is a discrepancy somewhere if they see many physicians driving luxurious cars, taking expensive winter vacations and showing other evidences of collecting considerable money from the public, when the hospitals from which they derive their incomes are having such a serious struggle. The public is not favorably impressed by signs of great wealth in a group whose tradition is to render service to those who are in need. Therefore it behooves us to recognize that we are public servants and not be too grasping.

What are the basic services that the public has a right to expect from physicians? As consumers they certainly have the right to expect certain things if they are to remain satisfied with our system of providing medical service.

They should expect to be able to find a doctor promptly when they call one. This means that all of us should have some definite system of being covered by associates when we are necessarily out of reach. It also means that as a society we should have some central method of answering emergency calls, instead of leaving it to the telephone company to set up its own list. This problem is being actively studied at present.

They should expect that doctors will continue to promote the advancement of medical knowledge through research and that all doctors will keep abreast of this new knowledge by attending staff meetings, taking graduate courses and keeping up with the literature.

They should expect that as long as the individual fee-for-service basis of payment continues physicians will take into account the economic situation of the patient, will discuss beforehand the fees when any large outlay is involved and will be fair in their charges.

They should expect that physicians, especially those in hospitals, will police themselves and exercise supervision over the work of those appointed to these staffs, so that when a patient goes to a hospital he can be sure of being treated by someone experienced in dealing with his particular condition.

To accomplish these things all physicians must have, in addition to technical training, a sense of social responsibility, which is largely determined by his own conscience and by his capacity to appraise his own abilities and recognize his limitations. Much can be done to keep each of us from getting out of line by means of participation in the various activities of our state and district medical societies, which still have an important function in spite of the growth and activity of hospital staffs and various special scientific organizations. Our society should be the focal point of our common contact with the public, and through it we should adjust our policies to be sure that we are meeting our social responsibilities at all times.

Since I have been recently serving on a local board of health I have been exposed to the public-health point of view as expressed in the policies of the American Public Health Association. A comparison of their objectives as expressed as recently as 1940 with those expressed in 1949 shows how far the public-health personnel have moved from the field of communicable diseases into the realm of individual noncommunicable diseases that affect large numbers of people, which we think of as clinical medicine — the province of the individual practitioner. Should this trend disturb us? What should be our attitude toward multiple-phase screening, health centers and so forth?

Only one criterion should be used in judging these services: What is the quality of the service rendered, and is it the best for the public? It has often been argued that physicians do their best work on a fee-for-service basis. That argument leaves me rather cold. A physician of high quality will give his best no matter what the method of remuneration. During the last war at least one third of the members of our profession were on salary. Both the record and my own observations show that these men did a magnificent job. Others have said that this was a temporary situation that would not hold for a longer pull. Now we see thousands of doctors working for the Veterans Administration and the Public Health Service — doing clinical work on a salary basis and rendering the highest quality of service. I myself have spent two four-year periods on a salary basis, and I do not think that there was any less incentive to good work.

The quality of the service is the only thing that counts. I do agree with the argument that we must protect ourselves against exploitation and subjection to demoralizing bureaucratic regimentation. We clinicians must not allow ourselves to be maneuvered into the inferior position of being mere technicians working under the direction of public-health or other officials who have not been trained in clinical medicine, but we must recognize that the consumers have a right to a voice in the management.

If we wish to preserve our present system of individual practice we must render a quality of service that justifies its continuation. By devotion to duty, by working together and teaching each other, by constantly raising our standards, exercising self-discipline and organizing self-supervision, we can justify our type of practice by the quality of service we render. In my opinion, it will be many years before any bureaucratic system can provide the continuous, individualized family service (including house calls at any time of the day or night) that the private practitioner now gives, with his accumulation of intimate knowledge of each family's needs.

Our society can and should serve as the agency for discussion and consultation, to ensure that we are rendering the best service of which we are capable.

As I approach my conclusion, I should like to quote from an address by Dr. John Romano delivered in 1948 before the Buck Hills Conference, from which my title was drawn.

> The function of medicine is to acquire knowledge and to apply it to promote health, to prevent disease, to free man and to help man to free himself from the enslavement of pain, illness and physical disability, and from the equally great enslavement of morbid fear, guilt, and neurotic personal and social restriction, in order that man can function to his maximum capacity as a conscious, intelligent, rational, free human being.

This may seem a very broad definition of the function of medicine, but after all, should we not have a broad definition of our aims and aspirations? If we limit our aims, we will become limited little persons. I should like to point out that under this definition there is a possible overlapping with some of the functions that we associate with the ministry. In this way we show our close relationship to the priesthood, from which we are, after all, descended. In ancient days the priest-doctor was one person. In rare instances even now a person is trained for both functions. In the more recent past there was a tendency to confine ourselves to the purely physical, or to heal illness purely by physical means, but now, largely through the influence of modern psychiatry, we again recognize that the physical and spiritual cannot be completely separated.

Furthermore, it would be well if we, in the spirit of the definition given above, applied to ourselves the old saying, "Physician, heal thyself." I refer

particularly to the latter portion of the definition concerning the enslavement of morbid and neurotic fear. Let us recognize that if we are torn by fears and suspicions we cannot, either individually or collectively, hope to free others from them. Let us cast out our fears of each other, of our government, of our future. Then we can stand as free individuals and play our full part, unhampered and unfettered, in this troubled world, which cries out for men of vision to cure its ills and heal its wounds.

MEDICAL PROGRESS

TREATMENT OF FRACTURES AND DISLOCATIONS

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With a Note on Anesthesia by

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THE last report on progress in management of fractures and dislocations that appeared in the *Journal* was written by Dr. G. W. Van Gorder in 1939.[1] He discussed the notable advances in this branch of surgery made since World War I. Several of the methods of treatment he mentioned have stood the test of time and can still be discussed under "Progress in Fracture Treatment," particularly Russell's traction for femoral-shaft fractures and the phalanged nail of Smith-Petersen for fractures of the femoral neck. To these must be added the improvement and standardization of metals for internal fixation; the intramedullary rod for long-bone fractures, especially the femur; and more effective management of compound fractures — all of which have been enhanced by perhaps the greatest advance of all, the discovery and extensive use of drugs to control infection, to say nothing of the employment of whole blood in supporting the severely wounded. War always gives impetus to the handling of all traumatic conditions; hence, much of our advancement has been made possible by extensive experience with the wounded in two world wars, not only in the immediate treatment of the local injury but also in the rehabilitation of the patient as a whole.

In the following pages we shall discuss briefly many factors that we believe have contributed to better management of skeletal wounds. We shall also try to give an over-all picture of present-day methods of handling the more common injuries of the long bones, the joints, the spine and the soft tissues related to these.

ORGANIZATION OF A FRACTURE CLINIC

Advances have been made in the teaching of fractures by means of organization of fracture services and fracture follow-up systems. Fracture services have functioned in a number of teaching hospitals for years, but it is only during the past two decades that they have been instituted in many of the larger clinics in this country. The organization of a specific fracture service and centralization of the fracture patients in a definite section of the hospital have given stimulus to the interest in and care of these patients. Staff rounds and visits are better organized, teaching is improved and nursing care can be carried out by more efficient standard routines. There is a distinct advantage in having the fracture service consist of those members of the general surgical service interested in trauma and its complications, the plastic service and the orthopedic service. Meeting regularly, the staff reviews all fractures and decides on a general policy of fracture management.

Dr. E. A. Codman[2] first proposed the idea of a follow-up system at the Massachusetts General Hospital in 1914, and follow-up studies of fractures were instituted there by Dr. Robert B. Osgood and Dr. Charles Scudder in 1919. Effort is made to re-examine every patient one year after injury. Routine x-ray films are taken and the patient is evaluated anatomically, functionally and economically.

INSTRUMENTS AND EQUIPMENT

It has been possible to treat an increasing number of fractures with open reduction and internal fixation with metal, due to two factors: improvement in surgical technic on the part of the surgeon and standardization of metals that are inert in living tissue. The two metals in common use are:

Stainless steel 18-8 S.Mo., of the following specifications[3]:

Chromium	17-20%
Nickel	10-14%
Molybdenum	2-4%
Manganese	2% maximum
Carbon	0.08% maximum
Silicon	0.75% maximum
Phosphorus	0.03% maximum
Sulfur	0.03% maximum
Rockwell hardness	30 to 35 C.

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Vitallium, developed by Venable in co-operation with Austenal Laboratories, which has been used as successfully as stainless steel, and is preferred by some surgeons. It contains the following:

Cobalt	65%
Chromium	30%
Molybdenum	3%
Manganese, silicon, carbon	2%

Some surgeons have used vitallium and 18-8 S.Mo. stainless steel in the same fracture without harmful results.[4] Never should any form of metal be used that will corrode in the tissue. One faulty screw is sufficient to cause marked local reaction, loosening of the fixation material and pain in the area.

In 1948, Eggers[6] described the slotted plate that was used with the expectation that muscle contraction will cause impaction at the fracture site. This principle has been illustrated by Eggers[6] in his work on bone healing in dogs' skulls, in which compression forces had been applied. Another type of slotted plate is the Collison type used by Peterson,[7] who applied two slotted plates of six screws each at right angles, in femoral fractures. Townsend and Gilfillan[8] have designed a plate that can be adjusted to length and impaction forces. Three types of intramedullary rods have been developed: the "cloverleaf" and U-shaped rods of Kuntscher,[9] the diamond-shaped solid rod of Hansen and Street,[10] and the curved Rush rod.[11] Notable improvements have been made in the design of tongs for skull traction. Those in general use are the Crutchfield-type tongs,[12, 13] the Vinke tongs[14] and the Barton tongs.[15] The Rogers extension frame[16] has been a singular advance toward easier care of skull-traction cases, as has the Stryker[17] rotation frame. Changes and improvements have been made in Balkan frames. The majority of the Balkan frames produced today are of metal, but a number of hospitals still use the cheaper and lighter wooden frames. Fracture beds have had the addition of many mechanical adjustments to alter the position of patients in bed. Fracture tables have also been improved. The quality and composition of plaster of Paris have been improved and better standardized.[18] Although plaster of Paris answers the need of most external fixation, the use of Castex and fiberglass has certain indications.

Anesthesia

As anesthesia continues to make use of the recent advances in knowledge, drugs and technics and well trained anesthetists are located throughout the country, we are better able to cope with particular problems in anesthetic management. Anesthesia for the emergency treatment of fractures is one of the most perplexing.

For the adult who has eaten prior to operation regional anesthesia is generally used, that is, brachial-plexus,[19] elbow and wrist block for the upper extremity. Reduction of Colles fractures under brachial-plexus block has proved outstandingly useful.

By and large, children still require general anesthesia. Stomach-tube aspiration or induction of vomiting should be considered imperative. *It must be emphasized that normal gastric emptying can be considered to have taken place only from the time of the last meal to the time of injury, — not since injury.*

Because of the duration and muscular relaxation required of most fracture procedures, ether remains the anesthesia of choice, although the safety of pentothal-nitrous oxide-curare is being explored.[20, 21]

Fractures of the Long Bones

Fractures of the Femoral Shaft

Harris[22] has stated that "Fractures do not lend themselves to complete regimentation as in the case of other fields of surgery, because they differ profoundly one from the other even in the same bone. Therefore, what is justifiable in the hands of a trained surgeon in one place in a properly equipped hospital is entirely unjustified elsewhere."

This statement applies to all long-bone fractures, and especially to the femur. In other words, the skilled bone surgeon is justified in treating many femur fractures by operation. However, the majority of such fractures occur near, and are treated in, smaller community hospitals where the attending surgeon may not be specially trained. In such locations, manipulation and plaster fixation or traction may be the safest methods.

The transverse displaced fracture may be amenable to manipulation and fixation in plaster spica. The oblique or comminuted shaft fracture will respond to traction.[23, 24]

In proper surroundings and in the hands of adequately trained surgeons,[4, 7, 24a] the use of plates and screws — and of bone grafts when indicated — has brought good results. It remains to be seen what place the intramedullary rod will assume in the management of femoral fractures. Traction is still the safest method, but this must be undertaken only by a surgeon with reasonable experience in and knowledge of the mechanical principles involved. Russell's traction[25] is one of the great contributions of this century to fracture treatment, and it is very effective in handling all shaft fractures above the distal quarter of the bone. Skeletal traction, preferred by some, may be more efficient if used with the Kirschner wire. Usually it should be placed through the tibial tubercle. This form of traction does not harm the knee joint and if placed in the tibia gives much more effective muscle pull, as the muscle attachments are at the upper end of the tibia.

Fractures of the Femur in Children

Fracture of the femoral shaft in children is a problem quite different from that presented in the adult. Below the age of twelve or thirteen, operative

treatment is rarely indicated. Under five or six years of age, Bryant's[26] overhead suspension of both lower extremities remains the method of choice. From the age of six to twelve years or older, Russell's traction will produce realignment and a successful result in a high percentage of cases.

Fractures of the Tibia and Fibula

Relatively few fractures of both bones of the leg with marked displacement can be effectively treated by closed methods. The decision to operate should be based almost entirely on the nature of the fracture, and also on the ability of the operating surgeon. Generally speaking, the transverse fracture that can be manipulated and locked in position can be managed in plaster fixation. The oblique fracture will usually become displaced if treated in plaster and must be handled either with skeletal traction (Kirschner wire in heel) or by operative means. If the fibula is intact, the oblique fracture can probably be managed with plaster alone. The badly comminuted fracture may not be amenable to open reduction and may heal better with skeletal traction for four to six weeks, followed by plaster fixation. The time of immobilization will vary greatly with individual patients, but as a rule the transverse fractures in good position can bear weight in a walking boot in six to eight weeks; the oblique or comminuted fracture, in eight to ten weeks. If open reduction and fixation have been done in the latter, — and particularly if bone grafts have been added, — weightbearing can be started perhaps two to three weeks earlier.

In the well organized fracture clinic, with experienced bone and joint surgeons, open treatment of both bones of the leg has become increasingly popular, with considerable justification.[4] Various methods of internal fixation can be used, according to the nature of the fracture; that is, the transverse fracture is best held with a 4-hole or 6-hole plate and screws. The oblique or comminuted fracture may be sufficiently held with two or more screws, although some surgeons may prefer a plate and perhaps also a transfixation screw. The Kuntscher rod in the tibia has not met with general favor in America, although some writers believe it has a definite place in management of these fractures.[27] It is rarely that plaster fixation is not needed after open operation. One should not rely on internal fixation in tibial fractures, and external fixation should be maintained until consolidation has advanced to a reasonable degree. Much, however, depends on the perfection of technic employed in applying the internal fixation.

Fractures of the Humerus

The majority of humerus fractures can be cared for adequately by closed methods of traction or splinting. One notable contribution of humerus-fracture management has been the hanging cast, as advocated by Caldwell.[28] Although its application seems illogical, it does bring about successful results in many cases, particularly in oblique fractures of the middle and lower thirds.[29] It is not applicable for surgical-neck or upper-shaft fractures.[30] The surgical-neck fractures are usually not grossly displaced and can be best handled by means of early active exercises initiated within a few days after injury and continued for many months afterward. Often the upper-shaft fractures will be displaced. Usually the distal fragment is pulled inward by the pectoralis major muscle. This type is best treated by keeping the arm at the side with a large pad in the axilla. Transverse fractures of the upper shaft without great displacement may best be managed by application of the plaster spica. Operative treatment is indicated when manipulation will not realign transverse fractures, when the surgeon suspects interposition of muscle between the main fragments of an oblique fracture, or when there is a widely separated "butterfly" fragment. The most useful approach to the humeral shaft is that advocated by Henry,[31] which follows the course of the cephalic vein and allows adequate exposure of the entire shaft without damage to important structures. For internal fixation of the transverse fractures the standard 4-hole vitallium or stainless-steel plate with appropriate screws may be used. The slotted plate of Eggers[5] would be useful in this type of fracture. With the oblique fracture or with the free "butterfly" fragment the use of screws only, without a plate, may be sufficient. The screws must be sufficiently long to engage the opposite cortex; usually three or more should be used. Many clinics routinely add bone grafts to openly reduced humeral fractures, either from the ilium or from a bone bank.

Fractures of the Forearm

Of all shaft fractures, those of the bones of the forearm call for the most accurate replacement, not only to produce union of bone but also to preserve function, particularly in pronation and supination.[32, 33] An analysis of the deforming factors after injury is important. For instance, fractures of the radius below the attachment of the biceps and supinator muscles and above the origin of the pronator radii teres will cause supination and flexion of the upper fragment and pronation of the distal fragment.

Forearm fractures call for early reduction under brachial block or general anesthesia. Manual traction followed by manipulation and plaster fixation may solve the problem of the transverse fracture, but the comminuted or oblique fracture will probably not hold in any form of external splint; therefore many of these cases will be better treated by open reduction and internal fixation. Prolonged skeletal or skin traction has been almost abandoned for treatment of forearm fractures, as have all forms of splinting except plaster of Paris or some of the cellulose substitutes.

Intramedullary rods can be used successfully in the ulna, but are not so suitable for the radius.[34, 35]

Forearm fractures in children present problems different from those in the adult. The greenstick fracture of one or both bones is very common. The resultant angulation must be corrected if necessary by fracturing the cortex and periosteum on the concave side. *Recurrence of deformity during the first few days following reduction must be guarded against by roentgenograms as necessary and by keeping the plaster splints sufficiently snug to offset diminution of swelling and development of muscle atrophy.*

Injuries to Joints

Greater emphasis is now placed on the initial evaluation of the injured joint. An analysis of the nature of the injury and a careful local examination of the joint, under anesthesia if necessary, to determine the extent of damage not only to the bones but also to the ligamentous support and the articular cartilage, are important.

Such care on the part of the examining physician may lead not only to more adequate early treatment but also to a more accurate and a more favorable prognosis. Early detection of severe ligament damage may be an indication for immediate operative repair[36] or prolonged fixation in plaster of Paris.[37] Many severely displaced joint fractures, particularly those of weight-bearing joints, require accurate replacement, by open operation if necessary,— but each case must be evaluated individually and treated accordingly.

Carpal Injuries

Carpal Navicular

Every "sprained" wrist should be under suspicion for fracture of the carpal navicular. Routine anteroposterior and lateral views of the wrist may not demonstrate a fracture line in the navicular; thus it is necessary to take films in special positions.[38] A light forearm plaster cylinder, including the thumb, if worn for ten to twelve weeks will produce bony union in the large majority of cases. Nonunion occurs because of failure of early diagnosis and adequate fixation. Gordon Murray[39] found that bony union occurred in 96 of 100 old fractures after bone graft. Bone grafting is the treatment of choice for nonunion, unless there is arthritis of the wrist joint or an avascular necrosis of the proximal fragment. Sutro[40] has fused the navicular to the capitate in selected cases.

Lunate

Dislocation of the lunate represents the second commonest carpal injury. This dislocation is nearly always toward the volar side of the wrist and if seen early can be reduced by manual traction and manipulation. Aseptic necrosis of the lunate (Kienbock's disease) rarely occurs after dislocation. When present, however, symptoms may respond to a leather wrist support. If this fails, excision of the lunate bone is indicated.[38]

Capitate

Retrolunar dislocation of the capitate bone is always associated with fracture or rotation subluxation of the navicular.[41] Early manipulative treatment is indicated; if this fails, operation by the dorsal approach should be carried out. If the continuity of an associated navicular fracture is not restored, introduction of an autogenous bone graft should be done.[38]

Colles Fractures

The technic for reduction of Colles fractures, as first demonstrated by Rose,[42] is being more generally adopted. The method is simple and effective. The patient is prone, the arm secured with padded webbing and the elbow flexed to 90°. Traction is applied to the thumb, or thumb and fingers, from a pulley at the foot of the table. Usually 10 pounds' weight applied for ten minutes will reduce the fracture. Manipulation can be carried out effectively and the plaster support applied before the traction is released. The plaster gutter splint,[43] which is moulded over the radial aspect of the arm, allowing motion of the fingers and thumb, has proved very satisfactory in maintaining reduction. The elbow can be included in the cast, if necessary, for more adequate immobilization of the wrist. In some clinics, Kirschner-wire traction through the first metacarpal has been found necessary to maintain reduction of the severely comminuted Colles fractures.[44] In the elderly patient, extremely comminuted Colles fractures have been treated with only an anterior splint support, allowing complete use of fingers and thumb.[45]

In the young person, the malunited Colles fracture may call for operative procedure, either osteotomy of the lower end of the radius[46] or resection of the distal end of the ulna.[47]

Fractures of the Elbow

Supracondylar Fracture in Children

The most common elbow injury in children is the supracondylar fracture. This is followed in frequency by fractures of the lateral and medial condyles. McDonnell and Wilson[48] report poor results in 25 per cent of 53 supracondylar fractures in children. They warn of the complications that may follow this fracture when the fracture line extends through the epiphyseal plate. Early fusion of the epiphyseal plate may occur, with resultant slow but progressive cubitus valgus or varus. The fracture may heal and function return to normal, with this deformity developing years afterwards. As a general rule, however, when there has been adequate reduction and this has been maintained with the forearm in supination and the elbow in flexion, this fracture should give little

trouble. If reduction has not been maintained, one of two procedures may be followed: either Dunlop's traction[49, 50] or skeletal traction through the olecranon.[51] Open reduction is seldom indicated in children for this fracture.

Fractures of the Lateral Humeral Condyle in Children (Capitellum)

This injury may be followed by growth disturbance in 40 to 50 per cent of cases.[48, 51, 52] End-result figures are better in cases treated by open reduction,[48] but care must be exercised to preserve the blood supply of the epiphysis, which is supplied through its capsular and ligamentous attachments.

Fracture or Displacement of the Medial Humeral Condyle in Children

Displacement of the medial condyle does not result in as high an incidence of poor results as does that of the lateral condyle. Nonunion may occur, but growth disturbances seldom follow. The nonunion is usually asymptomatic, although later ulnar neuritis is a possibility.[53] Patrick[54] has described a manipulation to reduce the medial condyle that has been drawn into the joint. If this is unsuccessful, open reduction should be performed as recommended by Aitken and Childress.[55]

Fracture of the Neck of the Radius in Children

This fracture usually occurs distal to the epiphyseal line of the head. When closed manipulation is unsuccessful, open reduction is indicated.[56] Removal of the radial head is contraindicated, as cubitus valgus and disturbance in growth of the elbow will develop. If the epiphysis of the radial head has been displaced and closed manipulation has not replaced it, surgical replacement is recommended.[44, 57]

Fractures of the Elbow in Adults

In severe T-fractures, early motion of the elbow is important. Reduction usually can be obtained by overhead traction, with Kirschner wire through the olecranon[58] or, in cases where there are good fragments, open reduction[59] and internal fixation with either "Y" plate and small screws or Kirschner-wire internal fixation are effective ways of securing reduction and allowing early motion.[60] In severely comminuted compound fractures of the elbow, Nicholson[61] recommends careful débridement of the shattered bone fragments in order to avoid ankylosis of the elbow joint.

Fracture of the Radial Head in Adults

The majority of fractures of the radial head can be treated conservatively with a sling or plaster support. The joint may be aspirated if there is extreme pain due to hemarthrosis. Speed and Smith[62] list the indications for surgical intervention. The badly comminuted fracture requires total excision of the head and neck and should be performed early. Smith and others[63] review the occurrence of myositis ossificans from this injury and cite laceration of the joint capsule and hemorrhage in the brachialis muscle as its causes.

The Capitellum

Smith[51] reports resection of isolated fractures of the capitellum treated with early motion followed by good results. This fracture is subject to aseptic necrosis, whether treated conservatively or by open reduction.

Fractures of the Olecranon

Fractures of the olecranon with displacement in adults have been treated by open reduction with various means of internal fixation. These include threaded wires,[64] stainless steel wire suture,[65] small Smith-Petersen nail, fascia repair,[62] and vitallium lag screw.[66] McKeever and Buck[67] report that successful resection of olecranon fragments that constitute as much as 80 per cent of the olecranon is possible, provided that the coronoid and the vertical distal face of the trochlear notch are preserved. They report stable elbows and very early use. This method of treatment is particularly applicable in the older age group.

Dislocations of the Elbow

Dislocations of the elbow should be reduced as soon as possible and protected by a posterior splint or a circular cast in flexion of about 90° for a period of two weeks, after which exercises should be begun. Associated fracture of the coronoid process may occur and would require longer immobilization. The problem of old unreduced dislocations of the elbow is well reviewed by Smith and Speed[62] and Van Gorder.[68] These dislocations are usually posterior, and ordinarily after three weeks they are regarded as irreducible except by surgery.

Monteggia Fractures of the Elbow

Evans[69] has recently presented a new concept of this fracture-dislocation, in those cases with anterior dislocation of the radial head. He states that the mechanism is a forced pronation injury and that reduction can be completed and maintained by full supination of the forearm. He demonstrated experimentally the mechanism of the injury in 18 dissecting-room specimens. He reports 11 cases, in 9 of which he was successful in reducing and maintaining the reduction in full supination. When open reduction is indicated, internal fixation of the ulna by a plate or onlay bone graft is sufficient.

The Shoulder

Fracture-Dislocations of the Shoulder

Many cases of fracture-dislocation of the shoulder require surgery for best results. Some cases are successfully reduced by closed methods when seen

early. At operation, anatomic reduction should be accomplished if possible. If the humeral head is not severely comminuted the internal fixation may be stainless-steel wire, Blount-type plate or lag screw. In some cases the Nicola technic for recurrent dislocation of the shoulder will suffice. Lawrence Jones[70] has devised a tendoplastic procedure for those cases in which the humeral head is badly comminuted.

Dislocations of the Shoulder

The incidence of initial anterior dislocation of the shoulder is higher in the sixth decade than the second; however, the incidence of recurrent dislocation is appreciably higher in the second decade and decreases sharply between thirty-five and forty years of age.[71] This has influenced the period of post-reduction immobilization. In the younger age group the shoulder is immobilized for four to five weeks, whereas in the fourth decade support to the shoulder is maintained only temporarily for comfort. There is much difference of opinion concerning the nature of the injury in recurrent dislocation of the shoulder[72-74]; however, the most consistent findings are those first noted by Bankart[75] in 1938. Bankart's method of repair, or some modification of his technic, has been extremely satisfactory over the past fifteen years.[76-80] The incidence of posterior dislocation is roughly 2 per cent.[71] Methods of repair are reported.[81, 82]

Fractures of the Clavicle

Undisplaced fractures of the clavicle or slightly displaced fractures can be treated very successfully with routine figure-eight support. In those cases of displaced fractured clavicles in which reduction is necessary to restore length to the clavicle or to reduce deformity, the method recommended by Wilson[83] of reducing the clavicle with the patient sitting or lying, with application of a light modified shoulder spica, is very effective. This is applied initially and need not be removed until union is firm. Gordon Murray[84, 85] and McKeever[86] have devised the simplest and most effective form of internal fixation of the clavicle by an intramedullary 3/32" stainless-steel wire.

Acromioclavicular Separation

In young persons, strain or incomplete tear of the coracoclavicular ligament responds well to strapping.[87] In adults, however, complete rupture of the coracoclavicular ligament usually occurs. This is best treated by open operation. Many methods have been proposed,[88-94] the choice of which is direct repair of the coracoclavicular ligament with silk and securing the clavicle to the coracoid with fascia lata and circumferential silk suture.

Sternoclavicular Separation

Open reduction and repair are indicated in complete dislocation in the young adult.[95-97] The method of Speed[92] is more commonly used. In cases of old dislocations with traumatic changes in the sternoclavicular joint, resection of the inner end of the clavicle, instead of replacement, provides the best result.[92]

(To be concluded)

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 37331

PRESENTATION OF CASE

First admission. A twenty-six-year-old woman entered the hospital because of cough, weakness and peripheral edema.

Six months prior to entry for the first time the patient noted swelling of the left ankle and dorsum of the left foot. There was no fever and there were no other joint symptoms. Two months later she complained of cough and chilly sensations of two weeks' duration. A physician found a red throat, a rapid heart rate and a "moderately loud systolic murmur at the apex." There was no fever. She seemed initially to respond satisfactorily to 3 doses of penicillin and cough medicine. Two or three weeks later, however, the cough was still present and she was febrile, had night sweats and had become dyspneic. A few basal rales were heard in the chest; the heart was unchanged; there was no edema or "cardiac enlargement." She was treated at home with aspirin, 10 gr. every four hours, and then with cortisone, 75 to 100 mg. per day. She also received liver and iron and terramycin. Despite these measures the patient developed progressively increasing dyspnea with orthopnea.

Three months before admission she was admitted to another hospital for two months. A few basal rales were heard again in the lungs. There was a "loud, rough, systolic murmur at the apex." The liver and spleen were not felt. The temperature was 101°F., the pulse 120 and the respirations 24. The blood pressure was 105 systolic, 70 diastolic. The urine was normal except for a few white cells in the sediment. The hemoglobin was 68 per cent, the red-cell count 3,500,000 and the white-cell count normal. The nonprotein nitrogen was 77 mg. per 100 cc., later falling to normal. The electrocardiogram showed flat T waves in Leads 2, 4 and 5 and inverted T waves in Lead 3. She was maintained soon after admission on digitoxin, penicillin and cortisone.

About two months before admission cortisone was discontinued. At this time she began to develop abdominal distention and swelling of the right arm and of the lower extremities, more marked on the right. She also occasionally coughed up blood-streaked sputum but had no definite chest pain. The symptoms continued unabated, with some nausea and vomiting, and the heart was thought to have enlarged slightly during the course. She was placed on a low-salt diet. Because digitoxin did not slow a persistently elevated pulse or improve cardiac status this was stopped one week later. She then developed severe peripheral edema, requiring mercurial diuretics. Six hundred cc. of fluid was removed from the right chest; a culture was negative. A sacral decubitus but no other skin lesions developed during the period of hospitalization. There were no known chills or hematuria. She was discharged unchanged. Three weeks later she had two attacks of nocturnal dyspnea and was admitted to this hospital. The temperature was intermittently normal and elevated as high as 100°F.; the pulse continued to be in the region of 100 to 120.

The patient had scarlet fever, measles and whooping cough as a child. She had had frequent sore throats during the winters, and a tonsillectomy was performed at the age of twelve. There was no history of joint pains, nosebleeds, abdominal pain, restricted activity or other complaints as a child. She had no known heart disease and did not have diphtheria.

Physical examination revealed a chronically ill, emaciated woman with a cyanotic tinge in the skin of the back and extremities. There was questionable early clubbing of the fingers. No petechiae or jaundice was present. The neck veins were distended and pulsating. There was a chest deformity with dorsal scoliosis and prominence of the ribs posteriorly on the right side. Signs of fluid were noted in the right chest. Examination of the heart showed the apical impulse to be diffuse almost to the left midaxillary line. There was a Grade IV and V apical systolic murmur and thrill with a diastolic gallop. A Grade III diastolic rumble was heard at the time of the initial examination but later was never noted to be louder than Grade I. The pulmonic second sound was increased. No other murmurs were heard, and there was no friction rub. The liver edge, felt 5 cm. below the costal margin in the midclavicular line, was tender. There was slight tenderness in the left upper quadrant, but no masses could be felt. There was flank and shifting dullness. The lower extremities showed +++ edema, more marked in the right thigh. The calves were slightly tender, more markedly so on the right.

The temperature was 98°F. by mouth and the pulse 130; the respirations ranged about 25. The blood pressure was 100 systolic, 88 diastolic.

The urine had a specific gravity of 1.020 and gave a ++ test for albumin; the sediment contained up to 25 white cells with clumps per high-power field but no red cells or casts. Examination of the blood revealed a hemoglobin of 12 gm. and white-cell counts ranging from 9000 to 15,400, with normal differential counts. The serum nonprotein nitrogen was 48 mg., the total protein 4.89 gm., with an albumin-globulin ratio of 1.8; the serum sodium was 126.9 milliequiv., the carbon dioxide 29.6 milliequiv., the potassium 4.8 milliequiv. and the chlorides 84 milliequiv. per liter. The prothrombin time was 34 per cent of normal. Two blood cultures showed no growth. The sedimentation rate was 1 mm. in 1 hour. The initial electrocardiogram showed a sinus tachycardia of 120 beats per minute and a right-axis deviation; the PR interval was 0.15 second, the QRS interval 0.07 second; the T waves were upright in Leads AVL, V_5 and V_6, flat in Lead 2, inverted in Leads 3, AVF, V_1, V_2, V_3 and V_4 and prominent in Leads 2 and 3. X-ray examination of the chest showed enlargement of the atrium and left ventricle, with elevation of the left main bronchus and left pulmonary artery. There was pleural fluid on the right side (Fig. 1).

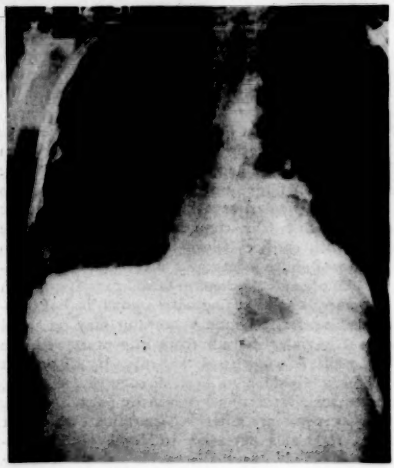

FIGURE 1.

The patient was maintained on routine cardiac measures, including mercurial diuretics and digoxin. One day after admission a thoracentesis yielded over a liter and a half of clear, straw-colored fluid with a specific gravity of 1.010 and a total protein of 1.8 mg. per 100 cc. There were 245 red cells per cu. mm. and 19 white cells per cu. mm., of which 11 were lymphocytes, 6 neutrophils and 2 monocytes. On this same day a chest film showed an area of density in the basal segments of the right lower lobe. At this time the patient complained of shoulder pain on the right side and produced some bloody sputum. Later on this day another x-ray examination showed the area of density to be clearing somewhat, and there was again evidence of a small amount of fluid on this side. On mercurial diuretics the chloride and the sodium levels fell. Two weeks after entry ammonium chloride was given intravenously, followed by a brief period of hypotension.

One month after admission she was started again on doses of cortisone, 50 to 100 mg. per day. She developed numerous petechiae and some ecchymoses. There were no chills or fever and little change in the course and she was discharged at the end of six weeks to a convalescent hospital.

Final admission (one week later). The course in the convalescent hospital was essentially the same as in this hospital. There was one episode of unexplained drop in blood pressure, with absent radial pulse or blood pressure and with apparently regular heart sounds of good quality, which seemed to respond to aminophylline. Two blood cultures were negative. She was maintained on digitoxin, a high-potassium diet and cortisone. No mercurial diuretics were given.

Physical examination was unchanged except for evidence of monilia infection of the mouth. The tongue was smooth and red. The blood pressure was 90 systolic, 60 diastolic. The temperature was 98°F., the pulse 120 and the respirations 28.

The blood sodium was 110 milliequiv. and the chloride 79 milliequiv. per liter and the nonprotein nitrogen 68 mg. per 100 cc. Because of the low sodium and chloride values ammonium chloride and hypertonic saline solution were given intravenously, after which she again became slightly hypotensive. Repeated determinations showed the sodium to be 122.7 milliequiv., the chloride 83 milliequiv. and the potassium 4.9 milliequiv. per liter and the nonprotein nitrogen 77 mg. per 100 cc. She was in shock, markedly cyanotic and unable to take anything by mouth because of nausea and abdominal pain. On the tenth day cortisone was stopped and penicillin started. The temperature rose to 101°F. for the first time during the entire course in the hospital, the pulse was 150, the respirations were 40 and the patient died quietly.

DIFFERENTIAL DIAGNOSIS

DR. HOWARD B. SPRAGUE*: This case, then, is that of a young woman without known previous heart disease who died in six months with an increasing cardiac enlargement and relentless heart

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failure without auricular fibrillation and with an essentially afebrile course, although she did have bouts of fever at times. The presenting signs were cough, dyspnea, orthopnea, edema, blood-streaked sputum and a loud apical systolic murmur. The positive laboratory findings were those of disturbed blood chemistry — low sodium, chloride and total protein and at times an elevated nonprotein nitrogen. Moreover, there was x-ray evidence of left-atrial and left-ventricular enlargement with elevation of the left main bronchus and left pulmonary artery and right-axis deviation evident in the electrocardiogram.

May I look at the x-ray films now?

DR. JOSEPH HANELIN: These chest films cover a period of time during the first admission to the hospital. Two sets of films were made on the day of admission before and after thoracentesis. The heart is quite large. There is distinct evidence of left-auricular increase, with elevation of the left main bronchus. The heart is enlarged toward the left; however, I would question that it is due principally to left-ventricular enlargement. The right ventricle appears enlarged and is particularly prominent anteriorly. Fluid is present in the right pleural cavity. After thoracentesis very little fluid remains. There is some streaky density within the basal segment of the right lower lobe. Rather interestingly, the pulmonary vessels are not particularly large in the peripheral zones of the lungs. A week later and at the time of discharge little change is noted from the last examination on the first day of the hospitalization. There is persistence of a small amount of fluid at the right base and also evidence of the enlarged liver and some degree of abdominal ascites, which were noted clinically. I can see no obvious splenic enlargement.

DR. SPRAGUE: Can you say anything about the main pulmonary artery?

DR. HANELIN: Although it is not huge it appears to be a sizable vessel. Particularly it appears to be rather large in contrast to the small aorta.

DR. SPRAGUE: Do you see any densities that would suggest tumor in the right lower lobe?

DR. HANELIN: No.

DR. SPRAGUE: It looks to me as though there were prominence of the vessels on the right side of the heart.

The immediate conclusion might be that this woman had fulminating rheumatic fever with mitral regurgitation. I am apparently in good company with this suggestion because she received cortisone, perhaps on the assumption that that was what had occurred. However, she had no joint involvement and no fever at the onset of this illness, and there was no response to cortisone. That seems to me to be a very unusual course for adult rheumatic fever, particularly in its first bout. There was no evidence that she had any heart disease before, although such evidence in the history is not always reliable — at least she had no limitation of activities. It does not seem likely that she had rheumatic heart disease prior to this illness, although it could not be disproved.

Secondly, was this an acute or subacute bacterial endocarditis with an ulcerative process of the mitral valve? I am rather impressed with the fact that this was a systolic murmur of considerable intensity, which was said to be at the apex. It is true that mitral regurgitation can result in intractable left-heart failure, just as can aortic regurgitation on the basis of ulcerative endocarditis. There was, however, little fever, no splenomegaly, negative blood cultures and insignificant petechiae. I suppose I cannot exclude this completely, any more than I can exclude completely a rare bacterial involvement of the pulmonic valve. It seems to me that she was not treated for subacute bacterial endocarditis — at least, that apparently was not the belief of the service — because she was not given massive doses of penicillin.

What other process may have involved the mitral valve? A rare condition such as tumor or spontaneous rupture of a cusp could do so. We had a case of spontaneous rupture of a mitral cusp in an older man last year at one of these conferences. The murmur in that case was believed by everyone to be that of calcific aortic stenosis. It does not seem likely that it would happen at this age. Could there have been an underlying congenital cause such as an interventricular septal defect or a subaortic stenosis? But why should the murmur become louder with cardiac failure in either of these two conditions? Such a murmur must indicate a considerable pressure gradient — blood passing through a relatively small opening with a good deal of pressure behind it. A Grade V murmur may be heard without having the stethoscope in contact with the chest wall. A murmur of that intensity, if present previously, would have been discovered medically, or even socially, during her healthy periods of life. If this were primary mitral regurgitation I would not expect right-axis deviation to develop under observation. This raises a question about the x-ray interpretation. Did this represent left-ventricular enlargement? Invariably, when the electrocardiographic findings disagree with the x-ray findings the electrocardiogram tends to be more reliable than the x-ray films. The T waves were inverted over the precordium on the right side. One would think of clockwise rotation of the heart and the sort of thing that occurs with right-ventricular strain, and there was constant sinus tachycardia. I do not know how much I can depend on the chest deformity to help me out of the difficulty I am in. Whether or not the chest deformity — scoliosis and displacement of the heart or perhaps rotation of the heart — modified the murmurs I do not know. Perhaps the turbulence that was heard and felt was not in the mitral valve but in the pulmonic valve, as the right

ventricle apparently comprised the whole anterior surface of the heart. The pulmonic second sound was increased, and it seems quite likely that the gallop rhythm was right ventricular, rather than left ventricular, in origin.

The onset of the patient's trouble coincided with the swelling of one ankle and the dorsum of the left foot. This suggests a thrombophlebitis as a background for the trouble. To support that diagnosis, later there was a swelling of the right arm and of the legs, tenderness of the calves, bouts of coughing up blood-streaked sputum associated once with shoulder pain, the area of density in the right lower lobe that cleared somewhat, leaving fluid, and the questionable early clubbing of the fingers. The unremitting downhill course of the disease suggests that the pulmonary circulation may have been involved more than other parts of the circulation. I find myself in a dilemma. There was a very loud apical systolic murmur, signs of what seemed definitely to mean mitral regurgitation and perhaps some mitral stenosis, but there were none of the other findings that I should like to have for the diagnosis of chronic mitral stenosis, such as a sharp first heart sound and the appearance of auricular fibrillation. I do know, for example, that the systolic murmur of aortic stenosis may at times be heard more loudly at the apex than in the aortic area. Could it be possible that some process in the main pulmonary vessels produced some degree of stenosis and was responsible for this? In other words, was the heart condition with mitral regurgitation the primary mechanism of failure and the pulmonary condition secondary, or was the pulmonary primary and the murmur put in here to make it more difficult?

So often I stand in these conferences like a swimmer about to take a cold plunge — and one has to come to some conclusion. I think this was a case of cor pulmonale with repeated pulmonary emboli from phlebothrombosis and secondary thrombi in the pulmonary vessels.

Clinical Diagnoses

Rheumatic heart disease.
Rheumatic fever.

Dr. Sprague's Diagnoses

Cor pulmonale.
Phlebothrombosis, with pulmonary emboli and secondary thromboses.

Anatomical Diagnoses

Acute bacterial endocarditis of mitral valve in a period of remission.
Rupture of chordae tendineae.
Infarct of lung, right upper lobe, recent.
Chronic passive congestion of liver, severe.

Pathological Discussion

Dr. Tracy B. Mallory: I did not have the advantage of seeing this post-mortem examination or even the gross organs in the fresh state, and the findings are so unusual and inconsistent that I particularly regret this. However we do have some excellent color slides of some of the organs, which I shall show you. The first one is the cut surface of the liver, showing a pattern of rather feathery or fern-like branching light areas against a dark reddish-bronze background, the characteristic picture of a very severe chronic passive congestion that has gone on to the point of considerable liver-cell necrosis and a focal regeneration. If the patient had been able to survive for any considerable period of time — as much as a year — the end result would have been a characteristic cardiac cirrhosis of the liver. There is as yet hardly enough increase of

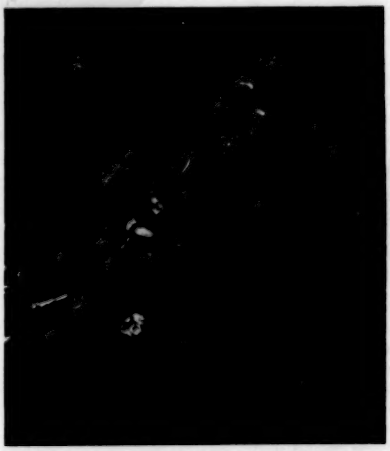

Figure 2.

fibrous tissue to justify using such a term. We can merely call it extremely severe chronic passive congestion with extensive liver-cell necrosis.

The next picture (Fig. 2) is centered on the mitral valve and shows, in the region of the valve and certainly on the chordae tendineae, yellowish excrescences that I am told were rather soft and putty-like and slightly gritty. A number of the chordae in the immediate neighborhood of these excrescences were ruptured. The valve leaflet itself is quite remarkably thin and transparent. One can see the underlying ventricular musculature quite easily through it. It is impossible for me to conceive of a rheumatic infection, either fresh or old, that could leave a mitral valve as thin and transparent as this one is, — so I think rheumatic endocarditis can be

ruled out. Moreover, rheumatic endocarditis would not be a satisfactory explanation for rupture of chordae tendineae. If the mitral valve showed even a slight degree of old scarring I think one could accept this gross appearance as evidence of subacute bacterial endocarditis. In the absence of any evidence of rheumatic involvement I believe this has to be eliminated. Also, the myocardium showed no stigmas of rheumatic infection. So I feel virtually forced to a diagnosis of acute bacterial endocarditis. Microscopical examination gave us no support for this diagnosis. No bacteria were demonstrable, and there was no significant degree of inflammatory reaction such as one would expect. Possibly the lack of inflammatory response can be explained on the basis of the various therapeutic agents, — chiefly the antibiotics, — that could conceivably eliminate the bacteria, although, as Dr. Sprague pointed out, more intensive therapy would probably have been required to produce a bacteriologic cure in such a case. We certainly know very little about what cortisone may do to inflammatory reactions in tissues. It can abolish many of the symptoms of infection, as Finland* showed in certain cases of pneumonia. In his cases the patients' fever cleared, as well as the malaise and sense of illness. As I remember, there was no significant change in the x-ray appearance of the lungs, and the blood cultures remained consistently positive. The patients were able to get up and walk around, although abundant pneumococci could readily be cultured from any specimen of blood that was taken.

Returning to our own case, the lungs were of some interest. In the lower lobe a scar was found, corresponding in location to the focus visualized by x-ray examination shortly after the first chest tap was performed. I would like to have found in this area an identifiable picture of an old infarction, but it did not appear in our sections, which showed only a localized fibrous thickening of several interlobular septa in this area. In the right upper lobe was a hemorrhagic focus with considerable necrosis consistent with infarction of several days' duration. However, the majority of the branches of the pulmonary artery were free from thrombi or evidences of sclerosis.

The spleen was essentially normal in size, as were also the kidneys. In cases of questionable bacterial endocarditis confirmatory evidence can often be obtained from examination of the kidneys, which characteristically show a focal glomerulonephritis often referred to as embolic glomerulonephritis, although the emboli are rarely if ever demonstrable. In this case none were seen.

Although I do not feel altogether satisfied about the diagnosis, this case seems to be more consistent with healed or at least quiescent acute bacterial endocarditis than with anything else.

*Kass, E. H., Ingbar, S. H., and Finland, M. Effects of adrenocorticotropic hormone in pneumonia: clinical, bacteriological and serological studies. *Ann. Int. Med.* 33:1081-1098, 1950.

DR. SPRAGUE: May this be an early chapter, then, in a new system of pathology — namely, disease as affected by steroid hormones? I think it may come up in other organs than the heart. It is very interesting.

DR. HANELIN: How large was the heart, Dr. Mallory?

DR. MALLORY: It was not large; it weighed only 300 gm. The cavities were moderately but not extremely dilated. The left auricle was perhaps the most strikingly dilated chamber in the heart. She was a very small and extremely emaciated person, so I think it is quite possible that a more significant hypertrophy may have been present at some time, part of which was later lost during the period of severe malnutrition.

CASE 37332

PRESENTATION OF CASE

A thirteen-year-old boy entered the hospital complaining of excessive fatigue and fever.

Twelve months before admission the patient began to have fatigue, a moderately sore throat, a tendency to bruise easily and low-grade fever. He was treated with penicillin at home, with subjective improvement for five or six weeks, when he was awakened from a sound sleep by sharp precordial pain and shortness of breath. These symptoms subsided spontaneously in a few minutes but recurred to some degree for the next two days. The patient was admitted to another hospital, where he was judged to be acutely ill. A systolic murmur was heard, but the heart was otherwise unremarkable. There were decreased breath sounds and dullness over the lower chest bilaterally. A chest film was said to be unremarkable and an electrocardiogram showed only sinus tachycardia. The sedimentation rate was elevated and "streptococci" were said to have been recovered from a blood culture. The patient was given a six-week course of penicillin and was discharged much improved after four months' hospitalization. He had a gradual weight loss amounting to 9 or 10 pounds.

The patient was kept on limited activity at home and had an uneventful course until four months before admission to this hospital, when he passed a small amount of bright-red blood in a bowel movement. A barium-enema examination was said to show no abnormality at this time. The patient continued to pass bright-red blood at approximately weekly intervals thereafter, usually well mixed with brown stool. Four weeks before admission the patient was permitted to return to school and almost immediately began to complain of severe fatigue. He also commenced to have 3 or 4 loose bowel movements each day, frequently flecked with bright blood. Five days before admission he had an ex-

acerbation of fatigue and noted a sore throat. He vomited once and had a small epistaxis. His physician found the temperature to be 100.2°F. and gave him penicillin, after which the temperature declined. He was then referred to this hospital.

There was no history of joint pains or swelling. The patient had been delivered at term and had normal growth and development. His only previous illnesses had been measles, mumps and whooping cough without sequelae. The patient's father, aged forty-three, had been an invalid for three years with rheumatic heart disease. An eighteen-year-old sibling had asthma. His mother and a nine-month-old sibling were living and well. The family had occasionally drunk raw milk, and one aunt had developed low-grade fever with abdominal pain. She had an elevated titer of brucella antigen, but organisms had never been recovered.

Physical examination on admission to this hospital revealed a thin but well developed boy in no obvious distress. The heart was not enlarged to percussion and had a normal sinus rhythm. There was a Grade I systolic murmur at the base that was loudest at the pulmonic area. The lungs were clear. The abdomen was soft, with normal tympany. The upper edge of the liver was percussed at the sixth rib and the lower border was easily palpable 2 cm. below the right midclavicular line and 5.5 cm. below the midsternal line. The edge was firm, sharp and not tender or nodular. The spleen and other abdominal organs were not felt.

The temperature was 99°F., the pulse 104 and the respirations 18. The blood pressure was 110 systolic, 70 diastolic.

The urine had a pH of 6.5 and a specific gravity of 1.015. It contained 2 white cells per high-power field. Examination of the blood revealed a hemoglobin of 7.6 gm., a red-cell count of 2,900,000 and a white-cell count of 23,500, with 81 per cent neutrophils, 9 per cent lymphocytes, 7 per cent monocytes, 2 per cent eosinophils and 1 per cent basophils. The reticulocytes were 1.2 per cent and the platelets abundant. The eosinophil count was 701 per cu. mm. The hematocrit was 32 and the corrected sedimentation rate 76 mm. per hour. The bleeding time was 7 minutes, the coagulation time 14.5 minutes. The mean corpuscular volume was 108 cu. microns, the mean corpuscular hemoglobin 30 micromicrogm. and the mean corpuscular hemoglobin concentration 28 per cent. A differential count of the bone marrow was within normal limits, with no tumor cells or abnormal forms seen. The nonprotein nitrogen was 28 mg., the total protein 8.54 gm., with an albumin-globulin ratio of 0.8, the cholesterol esters 69 mg., the alkaline phosphatase 15.3 units and the serum bilirubin 0.4 to 0.6 mg. per 100 cc. Cephalin flocculation was negative in 24 and 48 hours. The prothrombin time was 22 seconds (normal, 17 seconds), or 62 per cent of normal. The urine contained 0.2 units per 100 cc. of urobilinogen. The Congo red was 100 per cent retained in the serum. A blood Hinton test was negative. Agglutination tests for typhoid, brucellosis and tularemia were negative. Skin tests with old tuberculin were negative at concentrations of 1:1000 and 1:100. Four brown formed stools were guaiac negative. Two stools, which were watery brown grossly flecked with blood, were guaiac positive. Microscopical examination revealed many red and white cells but no ova or parasites. Stool cultures were negative for pathogens. An electrocardiogram failed to reveal any abnormality.

An x-ray film of the chest was negative. The spleen and liver appeared to be enlarged. A gastrointestinal series failed to reveal any abnormality of the esophagus, stomach or upper intestinal tract. A barium enema passed readily from the rectum to the cecum and refluxed into the terminal ileum. There was marked mucosal swelling of the entire colon and terminal ileum. In the transverse colon, descending colon and proximal sigmoid were many small ulcerations (Fig. 1).

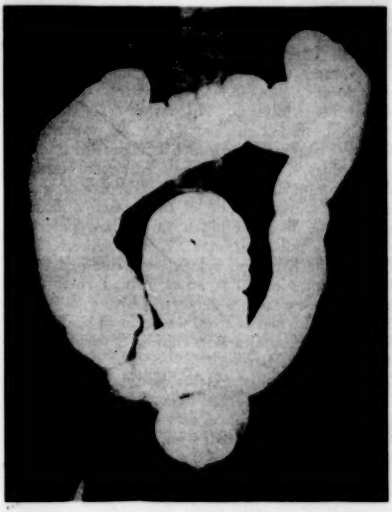

FIGURE 1.

Two sigmoidoscopic examinations performed soon after admission were essentially negative. A third examination revealed a coarsely granular friable area just inside the rectum that bled easily when wiped. The remainder of the bowel, which was visualized to a depth of 15 cm., was finely granular and friable.

The patient was in excellent spirits throughout the hospital stay. His appetite was good and he gained 3 pounds. He passed one or two stools each day; these were usually formed and unremarkable. The temperature ranged between 98 and 99°F. without specific therapy, and the white-cell count fell to 17,700. On the seventh hospital day an operation was performed.

Differential Diagnosis

Dr. Gertrud C. Reyersbach*: The symptoms with which this boy's illness began were consistent with a diagnosis of bacterial endocarditis. He had fever, fatigue, precordial pain, shortness of breath and weight loss. The other hospital evidently made that diagnosis after streptococci were cultured and treated him accordingly. But when one looks at the episode in retrospect there are several puzzling features. There was no history of congenital heart disease, and the findings on examination of the heart do not make me believe that the boy had congenital heart disease. His father had rheumatic fever, but the patient had no history or symptoms that were consistent with such a diagnosis. Bacterial endocarditis, if it is caused by *Streptococcus viridans*, is extraordinarily rare in the absence of a preexisting structural lesion of the heart. A bruising tendency is likewise much rarer than petechiae, which were not mentioned. I should like to know whether the liver and spleen were enlarged during the first episode and whether he had anemia. At that time he was considered to be very ill, as evidenced by the fact that he was kept in the hospital for four months and not allowed normal activity after he was discharged.

On his admission to this hospital the positive findings were an enlarged liver that was not nodular, definite disturbance of liver function as evidenced by an elevated total serum protein, an albumin-globulin ratio of 0.8, a normal total cholesterol value but diminished cholesterol esters, a prothrombin time of only 62 per cent of normal and a phosphatase possibly slightly elevated for a boy of that age. In addition, he had ulcerative colitis of the large bowel, as noted by sigmoidoscopy and by x-ray examination. The finding of white cells and red cells in the stool fits that diagnosis. Since the stool cultures were negative, I have to classify this as "idiopathic" ulcerative colitis. The anemia, which was macrocytic, fits in with liver disease, and so does the high sedimentation rate.

Before discussing this further, may I see the x-ray films?

Dr. Stanley M. Wyman: As recorded in the protocol, the lung fields are clear. The heart shadow is not grossly remarkable in size or shape. The esophagus, stomach, duodenal cap and loop show no localized abnormality. There does seem to be some prominence of the spleen. I am not so certain of the liver enlargement, but that also may be present. The films from the barium-enema examination show multiple fine spicule-like prominences along the margins of the colon that are particularly pronounced in the transverse and descending colon (Fig. 1). There is no alteration in the caliber or length of the colon. The films taken after evacuation of barium show diffuse swelling and prominence in the appearance of the mucosal pattern in the colon.

Dr. Reyersbach: Is that finding typical of ulcerative colitis?

Dr. Wyman: Not typical, but the fine multiple ulcerations are consistent with that diagnosis.

Dr. Reyersbach: Are there any films of the esophagus that show esophageal varices?

Dr. Wyman: The only films taken were the small spot films. The esophagus seems normal in caliber, and there is no definite evidence of varices.

Dr. Reyersbach: Some of the chronic diseases that this child may have had I think can be quickly ruled out by the negative tests and the clinical history; they are syphilis, tularemia and tuberculosis. As I read over the protocol, it occurred to me that the service might have been just as puzzled by this case as I am and that probably the physicians thought that one organ was abnormal and that if this organ was biopsied they might get an answer. I presume that the operation was a liver biopsy. My problem is to determine what the liver biopsy may have shown that helped to make a diagnosis.

There is little question in my mind that the child had ulcerative colitis; the sigmoidoscopic and the x-ray findings are entirely consistent with it. But children with idiopathic ulcerative colitis usually are not in good spirits, and this one was particularly described as being so. Children who have ulcerative colitis may develop cirrhosis of the liver, so it is possible that the two conditions may have gone together, but the ulcerative colitis was so mild and of such short duration that I cannot believe he developed liver cirrhosis secondary to that.

Another complication of ulcerative colitis is carcinoma of the large bowel. Usually it occurs later in the course, — and particularly with polypoid degeneration. There was no mention of polyposis in this case. Furthermore, the liver was described as smooth and not nodular, so I shall discard this diagnosis, too.

Primary amyloidosis is not described in childhood, to the best of my knowledge, and secondary amyloidosis with such mild ulcerative colitis I cannot quite believe to have been present. Amyloidosis seems to have been ruled out also by the negative Congo-red test.

Glycogen-storage disease may give symptoms both in the heart and in the liver, but usually the disease starts in early life. If the child survives, growth is stunted. If the first episode in our patient was due to this disease the boy should have had

*Associate physician to Children's Medical Service, Massachusetts General Hospital.

cardiac enlargement and cardiac failure. There are no laboratory tests that are specific for glycogen-storage disease. Glucose-tolerance tests and an epinephrine test, which are often abnormal, were not done. Most of the children with this disease have otherwise normal liver function. I should like to discard that diagnosis also.

That brings me to the diagnosis I should like to consider most seriously, — and with which I hope to explain all the child's symptoms except the elevated white-cell count and the increase in polymorphonuclear cells. The disease is brucellosis. The boy had ingested raw milk, and one member of his family was known to have had symptoms and to have had an elevated antigen titer. The first episode could have been the acute onset of brucellosis. The patient had fatigue and fever and was very ill at that time. We all know that it is very hard to culture the organism from patients with this disease. It has to be done on special media. Furthermore, the organism may look like green streptococcus in certain media, so I think it is possible that the organisms were cultured and were mistaken for green streptococci. Secondly, the profound fatigue was entirely consistent with chronic brucellosis, and the other symptoms that this child had are possible. The spleen may be enlarged. Enlargement of the liver occurs quite frequently, and organisms may be found in the liver; furthermore, cirrhosis is not an infrequent complication of chronic brucellosis. Very often there is weight loss, episodes of moderate fever and slight epistaxis. Constipation is much more frequent than bloody diarrhea, but the latter is not inconsistent with such a diagnosis. I admit that quite a few symptoms and lesions were missing. No glandular enlargement and no sweating were described. The agglutination test was negative, but this does not necessarily exclude the diagnosis. In about 10 per cent of the patients from whom the organism has been cultured the agglutination test remains negative. I would guess that the boy had chronic brucellosis, with secondary cirrhosis of the liver and ulcerative colitis.

Clinical Diagnosis

?Sarcoid, ?myeloma of liver.
Ulcerative colitis.

Dr. Reyersbach's Diagnoses

Brucellosis, with secondary cirrhosis of the liver and ulcerative colitis.

Anatomical Diagnosis

Portal cirrhosis of the liver, post-necrotic.
(Chronic ulcerative colitis.)

Pathological Discussion

Dr. Tracy B. Mallory: I think we might say at this point that Dr. Reyersbach was correct in her guess that the operation mentioned was a liver biopsy, — actually a needle biopsy, — so the amount of information that I can give you is going to be quite limited. Liver abnormalities are quite common in association with ulcerative colitis. However, they have proved very variable in type. Recently Kimmelstiel, Large and Verner* surveyed material from 90 fatal cases of idiopathic ulcerative colitis at the Armed Forces Institute of Pathology. They found significant liver abnormalities in 40 per cent. In only a small number was this frank cirrhosis. In many there was nothing more than nonspecific inflammatory infiltration.

The biopsy from our patient did show a frank cirrhosis, with complete distortion of the liver architecture and wide bands of fibrous tissue, which contained large numbers of proliferating bile ducts, — in other words, the characteristic picture of the post-necrotic type of portal cirrhosis. I can remember one patient treated here with an ulcerative disorder of the intestine associated with a diffuse fatty cirrhosis simulating the type we usually see associated with chronic alcoholism. That patient, however, had regional enteritis of the small bowel, rather than a chronic disease of the colon, as in this case. Another interesting case with probably some similarity of mechanism was one of cystic fibrosis of the pancreas associated with a markedly fatty cirrhotic liver. In ulcerative intestinal disease, deficiency syndromes of various types are not uncommon. It seems to be almost a matter of chance which particular dietary factor may fail to be absorbed in a given case and which deficiency syndrome may arise. We have seen in this hospital clear-cut evidence of Vitamin-A deficiency, of scurvy and occasionally of pellagra in cases of ulcerative colitis. One complicating factor that is often present in these patients is that in the course of the disease, especially when radical surgery was being contemplated, they have been treated with large doses of human plasma or with multiple whole-blood transfusions, so the possibility of homologous serum jaundice and hepatitis due to viral action must be considered. In this patient, so far as we know, there had never been either plasma or whole-blood transfusion therapy.

*Kimmelstiel, P., Large, H. L., Jr., and Verner, H. D. Liver damage in ulcer-active colitis. *Am. J. Path.* 26:715, 1950.

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A PARADOXICAL REFUGE

Science, by checking infectious diseases, has in a sense "created old age" as a phenomenon to be expected by the majority rather than the minority. However, along with this increase in life expectancy, the advancing years of many have been revealed as a period of life to be borne rather than enjoyed. Problems peculiar to old age have always existed, but only in recent years have they made a startling impact on society, — and then primarily only as a result of a rapid increase in the ranks of the aged.

Reliable figures demonstrating alcoholism's contribution to these problems are lacking. Inferences can be drawn, nevertheless, from what little evidence is available of the recorded deaths from alcoholism during the past decade, that 11 to 12 per cent were in the age group of 65 and above and 19 to 20 per cent were in the age group of fifty-five to sixty-five years. Alcoholism, however, is rarely reported as a primary cause of death, hence conclusions based thereon are likely to be untrustworthy. Of relatively more value are data based on first admissions to mental hospitals. Typical of the latter is the study made by Malzberg* in 1949, wherein 14 per cent of 1050 first admissions of patients sixty years of age and above to mental hospitals of New York State for the year ending March 31, 1948, were for alcoholic psychosis, with the psychotic symptoms being of relatively short duration.

On the whole, clinical experience indicates that the majority of elderly alcoholics first show evidence of their addiction at an earlier age, although a relatively large number first begin excessive compulsive drinking only after the age of sixty. Whether alcoholism is a carry-over from earlier years or originates late in life, the problem is a considerable one and is increasing with the aging population.

Three major factors tend to initiate or aggravate alcohol addiction in later years. First, there are changes in the body, — particularly in the nervous system, the endocrine glands and the general metabolism; secondly, there is increased vulnerability of personality structure, which is considered to be the resultant of inherited traits and early conditioning experiences; and thirdly, there are specific social and psychologic stress situations that are likely to develop at this age. Bodily changes and personality patterns not prepared to meet the vicissitudes and stresses of old age are predisposing factors that, when combined with specific physical, psychologic and social stress situations, can and do precipitate alcoholism.

Inefficient though it may be in its effect as such, alcohol is a depressant, a sedative and an analgesic. Furthermore, since it is readily available and generally both socially and culturally acceptable, many persons in painful situations turn to it for relief. Outstanding among these painful precipitating situations is that resulting from a progressively decompensating emotional adjustment. For instance, a person who has restricted his interests to

*Malzberg, B. First admissions with alcoholic psychoses in New York State, year ended March 31, 1948: with note on first admissions for alcoholism without psychosis. *Quart. J. Stud. Alcohol* 10:461-470, 1949.

one exacting field finds that he can be productive only up to a certain age and that he cannot easily shift his interests to another field. Thereupon an impasse is reached, and he resorts to alcohol as a means of allaying anxiety and relieving inhibitions so that he can carry on, at least temporarily.

Retirement with no previously anticipated or prepared alternative design for living may be so traumatic that support may be sought in alcohol. Moreover, in retirement, domestic tranquillity may be upset when a husband, having lost his empire at work, attempts to dispute, usually vainly, the control of the house, which has been and is the personal empire of the wife. Failing in his search for new empires, he turns to alcohol.

In later years, a lifelong protective environment or relation may break down, thereby exposing an inadequate personality to the full demands of reality. Such a typical situation occurs when a protective wife dies, leaving the dependent husband to manage affairs for which he has no capabilities.

In the treatment of elderly alcoholics there are several unique problems. Foremost is the fact that certain motivations that operate in the younger age groups of alcoholics are lacking: namely, hope for an expanding future, with restitution to society in good repute. Secondly, depth psychotherapy is difficult to carry out because of the elderly person's decreased capacity for reorganization. Finally, such adjuvants to treatment as the conditioned reflex and antabuse cannot be employed because of the cardiovascular impairments usually present. Therefore, treatment and rehabilitation for the most part must be based on psychosocial guidance.

Investigations of the physical changes and personality patterns that predispose to alcoholism will eventually lead to a decrease in the influence of these factors. However, of equal or even greater import are the psychosocial factors characteristic of advancing years that precipitate addictive drinking and militate against positive motivations for rehabilitation. Apparently social organization has not kept pace with medicine. Society has placed a premium on youth and at the same time neglected the old. Better medical care has resulted in prolonging life, but in addition to prolonging life, medicine has also rendered older persons more efficient physiologically and psychologically than in the past. Now society with the aid of medicine and its allied professions must study and establish ways and means not only of providing for but also of advantageously utilizing these new and hitherto unrealized capacities of the elderly. Moreover, traditions and attitudes must be changed if these added years are to bring an active and well adjusted life and not merely pain and frustration to be numbed by alcohol.

HOLIDAY MEDICINE

PRIOR to every holiday the National Safety Council issues a prediction on the deaths to be expected from traffic and other accidents. The holiday toll has assumed alarming proportions, although this year there was great improvement in this state over the Fourth of July. Our congratulations go to Registrar of Motor Vehicles King and his staff for this improvement.

Such figures, however, do not begin to cover the illness and disability that develop in the state and over the nation after week ends and holidays. Traffic accidents and water accidents account for a large proportion of these casualties, but picnics, work about the house and sports on land and water — all take their toll. Overexertion, carelessness, daredeviltry, food, drink, poison ivy, dermatitis and so forth make their contributions to the unhappiness of the next day and perhaps of many days thereafter.

The medical aspects vary from an acute medical or surgical emergency, with possibly a life at stake, to the mild disability that rest and time, proper hygiene or a simple home remedy will clear. The whole gamut of general practice may be covered in a single day. A gash on the arm, a sprained ankle, a baby with a headache and vomiting, a boy with severe pain in his belly, a punctured eye, a girl nearly drowned, an elderly man in a coma, an auto crash with three badly hurt and so on, may be the message when the telephone rings in the physician's office or in a hospital during the holiday period. A specialist would be cut of his depth in many cases, yet in some particular one his services might be invaluable.

Books on various emergencies have been written but it would take many pages, under the title of "Anything Can Happen and Its Treatment," to cover the whole range of the possibilities of holiday medicine. The physicians of resort places are the specialists in this field and are undoubtedly eternally prepared for anything. A physician deserves a holiday as well as anyone else, but as a part of their medical service to the community local groups of physicians in these resort areas should arrange to have the area covered around the clock. It can be a strenuously exciting life, — a test of preparedness, of skill and of versatility, — for the local physician in the minor to major unrehearsed, unexpected incidents that occur in holiday medicine.

WMA AND WHO

THE Fifth General Assembly of the World Medical Association will take place in Stockholm in September, at which time Dr. Dag Knutson, president of the Swedish Medical Association, will take over its presidency from Dr. Elmer L. Henderson, and matters of international import in medicine will be discussed by the assembled delegates. Some of them may even be settled.

Some confusion doubtless exists, in the minds of those who are only casually interested, in respect to the World Medical Association and the World Health Organization. Which does what and who is WHO? Actually, the relations between the World Medical Association and such agencies of the United Nations as the World Health Organization and the International Labor Organization have been a matter of some concern since the establishment of the WMA in 1946. To prevent overlapping, duplication of effort, misunderstandings and conflict, it is necessary that the role of WMA as an international organization of individual members of national medical associations be understood, and that WHO be recognized as an intergovernmental agency representing the various national health departments.

Much of this uncertainty was dispelled last May, when the council of the association, consisting of the representatives of twelve of the forty-one national-medical-association members, met in Geneva. At that time two-day conferences were held among delegates of the WMA, the WHO and the ILO to discuss such important problems as the improvement of standards of training for medical and related personnel and the points on which co-operative efforts might be made.

Six points were considered: the promotion of free flow of medical supplies between countries; the selection of suitable personnel for the far-flung activities of WHO; the "exchange of information and documentation on subjects of common interest," and contributions by each organization to the publications of the others; co-operation of WMA in securing the adoption of international nonproprietary names of drugs and other products, as recommended by WHO; the assistance of WHO in publicizing the "International Code of Ethics" adopted by WMA; and the representation of WMA in the regional conferences of WHO.

At the meeting in Geneva, also, the medical associations of Western Germany and Japan were accorded membership in the association.

— Down at Lockport they begin to write technical epitaphs thus —

The little hero who lies here,
Was conquered by the diarrhoea.

Boston M. & S. J., *August 20, 1851*

MASSACHUSETTS MEDICAL SOCIETY

ADDRESS DELIVERED ON MAY 17, 1950, BEFORE THE NEWLY FORMED SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

WALTER B. LANCASTER, M.D.

BOSTON

To be chosen to give the keynote address on the occasion of the founding of this Section is a mark of distinction that I appreciate as much as if I deserved it and that I accept with mingled feelings of pride and of humility.

Let me sound the keynote of our meetings — rather let me sound the major chord on which the harmony will be founded. The first note of the

chord is *enthusiasm!* Note the derivation of the word — ἐν θεος — a god within. As Pasteur exclaimed after thanking the Greeks for one of the most beautiful words in our language, "Heureux celui qui porte en soi un dieu!" Happy the man who bears a god within! Enthusiasm is the divine fire. It burns in every soul, but to attain its highest intensity it needs the contact of many. Compare the heat from one lump of coal with that produced by many in contact.

Enthusiasm is contagious; therefore let us expose ourselves freely to the virus, let us tolerate no quarantine, let us overcome even our natural repugnance to exuberant feelings, that by some means — by all means — we may fan the flame of that divine fire, that we may be found to harbor the god within. "Heureux celui qui porte en soi un dieu."

Enthusiasm is the urge toward *service* — the greatest of our aims as physicians. It is the urge toward *research*, the greatest of the benefits we can confer on our world. It is the urge toward good *citizenship*, which our president yesterday set up as our goal as members of our Massachusetts Medical Society.

No ship can reach port, no matter how skillfully and wisely it is navigated, unless it has motive power, — either wind without or engine within. Enthusiasm is the motive power essential to any progress, any attainment. It is enthusiasm that makes possible the steady grind of monotonous work when results are slow. It is enthusiasm that underlies that infinite capacity for taking pains that spells genius.

I would not belittle the part of the wise navigator who steers the ship; therefore I take for my second note of the chord *Truth*. Let it be our constant aim to seek for the truth, for more knowledge, more wisdom. Let us strive to sift out and reject error. Let this section stand for the highest grade of scholarship, with no tolerance for shams, though we have only sympathy for error and an earnest desire to help ignorance, which we all humbly admit we share. Let there be no snobbish superiority of greater wisdom when all are so far short of perfection.

Effective knowledge may be expressed as a fraction. It is equal to the number of true facts divided by the number of untrue facts. Each "fact" that is not true distorts all the facts that are true. This section will ever strive to increase the true facts and to scotch and bury the untrue facts.

There are many beautiful notes clamoring for place as the third note of the chord, but I must not prolong this into an overture. Let me take *Thoroughness* as the third note. Let me urge that it be our daily practice. How many of our mistakes in diagnosis must be laid to lack of thoroughness! How many of our operative complications! How many of our imperfect therapeutic results! Who among us has not had the humiliation of having a patient sent back from a consultant with the correct diagnosis, which we could have made just as well ourselves if we had only been thorough! It is doubly humiliating, for we not only fail in our diagnosis as doctors but we fail in our duty to our patient as a physician whom the patient trusted, to whom he committed his health believing he was choosing the best. When work becomes too pressing or our strength inadequate for thorough work it is our duty to restrict our practice to what can be done conscientiously and thoroughly. When the cause of lack of thoroughness is lack of enthusiasm, then we should seek to kindle the fire of enthusiasm by more contacts with others at this and other medical meetings and read the transactions and other stimulating literature.

The last note of the chord — the dominant note, the note with the wonderful harmonics that impart those elusive overtones, those characteristics that make up perfection of tone quality — the last note is *Honor.*

Let this section stand ever more and more inflexibly for high standards of honor — professional honor and — fundamental to all — personal honor! Let those qualities that mark the gentleman and that reach their flower in the ideal physician be our goal. Let us strive to be worthy of the great exemplars of our profession, that each of us may humbly say: "I, too, am a physician and a gentleman." Let the keynotes of our policy or, if you prefer, the planks of our platform as a section of the Massachusetts Medical Society, be Enthusiasm, Truth, Thoroughness, Honor; then we have the answer to the question some of us have asked: "What is there for me in a new section of Ophthalmology and Otolaryngology?"

Come to these meetings, expose yourself to the contagion that comes from contacts with others, kindle the fire in your soul and fan it to a flame.

Then remember always that it is not only a place to *get* something — to get enthusiasm, to get inspiration, to learn something: this section is a place in which you can give something, — and surely the world is so made that "it is more blessed to give than to receive."

"What," you say, "have I to give?" Much. You give by adding to our numbers. Enthusiasm is possible with small numbers, even one alone, but it is far more likely with larger numbers.

Then you have *ideas;* you have or should have *questions*. You have little variations in ways of doing things that others would be glad to learn about.

I believe that it is rarely that a man goes back to his routine after attending a medical meeting without gaining something, even if only the vacation effect of a temporary escape from his environment.

NOTES FROM THE MEDICAL EXAMINER

SUBDURAL HEMATOMA OF NONTRAUMATIC ORIGIN

The medicolegal investigation of sudden, unexpected or violent deaths due to intracranial hemorrhage results in such lesions' being classified as due to natural causes or trauma. Subdural hemorrhages are generally considered to be of traumatic origin. The finding of such hemorrhage necessitates efforts to establish the manner in which the trauma was received.

Recently we have had the opportunity of studying two cases of subdural hematoma that, because of their pathogenesis, did not follow the usual pattern of requiring further investigation concerning precipitating trauma. Each of these developed as the result of a rupture of an abnormal cerebral vessel, with hemorrhage directly into the subdural space.

Case Reports

Case 1. A 21-year-old nurse had been well until 6 months before death. Concurrent with a local outbreak of poliomyelitis she complained of left-frontal and occipital headache; her symptoms were attributed to poliomyelitis, although a definitive diagnosis was lacking. In the interim she was well. Without history of exertion on the day of death, she was seized with severe pain in the head, lapsed into unconsciousness and died within 20 minutes.

Figure 1. *Case 1. Tract of Rupture of Aneurysm Extending from Space Occupied by Aneurysm through Brain and Pia-arachnoid above Large Blood Vessel (left center).* (*x* 7.)

The scalp and calvarium were normal. Approximately 175 cc. of partially clotted blood was present in the subdural space of the left middle fossa and extended into the anterior fossa. It covered the superior and lateral surfaces of the left cerebral hemisphere. There was no subarachnoid hemorrhage. The arteries at the base of the brain were normal. There was a 4-mm. irregular defect in the meninges over the inferior lateral portion of the left temporal lobe.

The finding of such hemorrhage ordinarily dictates subsequent police investigation. It was necessary to gain, as far as possible, as many of the medical facts available immediately from the autopsy to aid further investigation. Therefore, coronal sections were made through the unfixed central nervous system. Fortuitously, one such section exposed a 1.5-cm.-wide saccular aneurysm of a branch of the middle cerebral artery, 0.5 cm. beneath the surface of the left middle temporal gyrus. There was no fresh hemorrhage in the surrounding cerebral tissue, except along a tract that extended from the aneurysm to the cortical surface and communicated with the subdural space in the region of the meningeal defect described above (Fig. 1). Microscopical examination of the pia-arachnoid, in the region of the defect described above and overlying the left temporal lobe, showed no inflammatory reaction or scarring. There was gliosis and scant hemosiderin deposition in the adjacent cerebral tissue. No recent hemorrhage was present except along the tract extending to the subdural space. The aneurysm showed degeneration of the elastica in the sections stained by Van Gieson's method and absence of the media. No arteriosclerotic change or inflammatory reaction was present in the sections of the aneurysm. The aneurysm sac was partially filled with thrombus.

Case 2. A 21-year-old nurse had no history of significant previous illness. Two days before death she complained of headache but continued to work. She was found dead in her room.

The scalp was normal, and no fractures of the calvarium were found. There was a recent partially clotted subdural hematoma approximately 135 cc. in size overlying the superior and lateral surface of the left cerebral hemisphere. No gross subarachnoid hemorrhage was present. In a sulcus extending to the superior surface of the left paracentral lobule was a dilated, tortuous segment of a branch of the anterior cerebral artery and veins (Fig. 2). A portion of this existed as a thinned-out cystic vascular sac. There was a fresh hemorrhage, 2 x 1 cm., in the white matter immediately adjacent to this, extending along the sulcus through the gray matter into the subdural space.

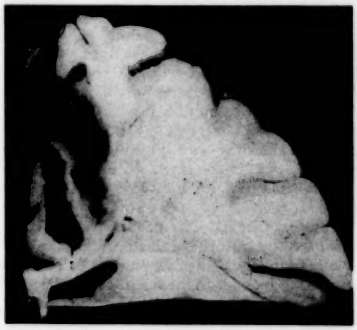

Figure 2. *Case 2. Vascular Abnormality on Medial Surface of Left Paracentral Lobule with Hemorrhage into Adjacent Cerebral Tissue and thence to Subdural Space.*

No evidence of previous injury to the meninges or cerebral tissue was found on microscopical study of the sections from this area. No inflammatory reaction was present in the vessel walls. The elastic-tissue stains showed the vascular abnormality to be of arterial and venous origin.

Discussion

The first case illustrates the congenital medial-defect abnormality of cerebral arteries described by Forbus.[1] Our general experience with such intracranial aneurysms parallels that of Robertson[2] and

Richardson and Hyland[3] concerning frequency of intracerebral hemorrhage with tissue destruction and intraventricular hemorrhage as opposed to uncomplicated rupture of such aneurysms into the subarachnoid space. That such aneurysms usually involve the arteries at the base of the brain in the anterior portion of the circle of Willis is appreciated. That they may involve the distal branches of the cerebral arteries has been stressed by others. To our knowledge, subdural hematoma has not been described heretofore as resulting from rupture of such aneurysms.

The second case represents a vascular abnormality of arterial and venous type, which on rupture resulted in subdural hematoma formation. Arteriovenous aneurysms, venous angiomas and secondary hemorrhage from meningocerebral cicatrix have previously been reported as precursors of subdural hematoma.

From the standpoint of medicolegal investigation it is important to appreciate such nontraumatic mechanisms of subdural-hematoma formation.

Summary

Two cases of nontraumatic subdural hematoma are presented. One of these resulted from rupture of a congenital medial-defect aneurysm.

The medicolegal importance of recognizing the nontraumatic origin of such hemorrhage is stressed.

References

1. Forbus, W. D. On origin of miliary aneurysms of superficial cerebral arteries. *Bull. Johns Hopkins Hosp.* **47**:239-284, 1930.
2. Robertson, E. G. Cerebral lesions due to intracranial aneurysms. *Brain* **72**:150-185, 1949.
3. Richardson, J. C., and Hyland, H. H. Intracranial aneurysms: clinical and pathological study of subarachnoid and intracerebral haemorrhage caused by berry aneurysms. *Medicine* **20**:1-83, 1941.

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BOOK REVIEW

Thoracic Surgery. By Richard H. Sweet, M.D. With illustrations by Jorge Rodriguez Arroyo, M.D. 8°, cloth, 345 pp., with 155 illustrations. Philadelphia: W. B. Saunders Company, 1950. $10.00.

It is always a pleasure to read anything by Dr. Sweet, because he has the faculty of clarity and directness of expression. So it is with his book; it reads easily and is interesting throughout. It is not cluttered with numerous references, and the author purposely states that he has omitted any bibliography because his intention is to write a book about thoracic procedures as he performs them, — with all of which he is familiar and in some of which he is a master. He has treated a very large subject with amazing succinctness. The anatomy and technic are extremely well illustrated by Mr. Rodriguez Arroyo. Essentially the book is on operative surgery, to help an already equipped surgeon to perform intrathoracic procedures. The section on the esophagus is the most complete in detail, although almost no mention is made here of the right-sided or combined approach to lesions in the upper esophagus. A short discussion might well have been provided, as some surgeons prefer this method of approach for such lesions situated in the upper third of the esophagus.

Dr. Sweet might perhaps write a different description and discussion of operations on the mitral valve for stenosis if he were writing his book today. This merely shows the rapidity with which change occurs in the development of surgery. For instance, instead of being in the doubtful zone, commissurotomy is now a well recognized procedure. These, however, are minor details in what is really a splendid book, which provides delightful reading because of its clarity and direct approach throughout.

Frank B. Berry, M.D.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Apuntes de cancerolojia para el prontuario del medico practico. By Dr. H. Pieter, Catedrático de la Facultad de Medicina de la Universidad de Santo Domingo consultante en el Instituto de Oncolojia "Milagro de la Caridad," de Ciudad Trujillo, R. D. 8°, paper, 66 pp. Ciudad Trujillo: Imprenta San Francisco, 1950. (Publicacion autorizada por la Universidad de Santo Domingo.)

The author presents in this pamphlet 285 aphorisms on cancer in general and on the various systems and organs of the body.

Newer Concepts of Inflammation. By Valy Menkin, M.A., M.D., associate professor of experimental pathology and head of the Department of Experimental Pathology, Agnes Barr Chase Foundation for Cancer Research, Temple University School of Medicine. 8°, cloth, 145 pp., with 67 illustrations. Springfield, Illinois: Charles C Thomas, 1950. $3.50.

This monograph represents supplementary data to the author's *Dynamics of Inflammation*, published in 1940. The four chapters represent four lectures delivered at a dental seminar in 1948. Bibliographies are appended to the various chapters; there is a good index, and the illustrations are excellent. The monograph should be in all collections on pathology.

German-English Medical Dictionary. By Joseph R. Waller, M.D., and Moritz Kaatz, M.D. New edition with supplement. 16°, cloth, 244 pp. Philadelphia: Blakiston Company, 1950. $2.50.

This pocket-size dictionary now in its seventh edition was revised by Dr. Braitenberg. The supplement comprises six pages of additional unusual words. The book should be in all medical libraries and should prove especially valuable to medical librarians and physicians translating medical German.

Antipyrine: A critical bibliographic review. By Leon A. Greenberg, Ph.D., associate professor of applied physiology, Yale University. With an introduction by Howard W. Haggard, M.D., director, Laboratory of Applied Physiology, Yale University. 8°, cloth, 135 pp. New Haven: Hillhouse Press, 1950. $4.00.

This monograph is No. 3 in a series issued by the Institute for the Study of Analgesic and Sedative Drugs. The previous publications were on acetanilid and the salicylates. The number of titles listed is 1735, and they are critically evaluated in a number of chapters preceding the bibiography. There is an index of subjects, and the book is well published. It should be in all medical libraries and collections on drugs.

Basic Principles of Clinical Electrocardiography. By Hans H. Hecht, M.D., associate professor of medicine, University of Utah School of Medicine. 8°, cloth, 88 pp., with 32 illustrations. Springfield, Illinois: Charles C Thomas, 1950. $2.00.

Dr. Hecht, in this small book, discusses the unipolar semidirect leads, unipolar limb leads, and bipolar limb leads (the standard leads). A bibliography and a glossary of terms conclude the text.

Reprints from the Oxford Loose-Leaf Medicine. 8°, paper. New York: Oxford University Press, 1950. (Not for sale.)

The Oxford University Press has reprinted nine articles from the *Oxford Loose-Leaf Medicine* for the benefit of subscribers of the system. They include the *Autonomic Nervous System*, by Keith S. Grimson, *Encephalitis and Other Virus Infections of the Central Nervous System*, by Goronwy O. Broun, and the articles on ancylostomiasis, tetanus and gas gangrene, lesions of the cauda equina and conus medullaris, relapsing febrile nodular nonsuppurative panniculitis (Weber–Christian disease), mycosis fungoides, lymphoblastoma of the skin and allied conditions as general diseases, the clinical examination of the cerebrospinal fluid and psittacosis. These reprints are valuable to medical libraries for lending purposes, making it possible to retain the volumes of the set in the libraries.

The Clinical Use of Testosterone. By Henry H. Turner, M.D., clinical professor of medicine, School of Medicine, University of Oklahoma, Oklahoma City, Oklahoma. 8°, paper, 69 pp., with 9 illustrations. Springfield, Illinois: Charles C Thomas, 1950. $2.25. (Publication No. 46, American Lecture Series.)

This monograph is based on the literature of the subject. The introduction comprises a short history and is followed by chapters on chemistry, physiology, the metabolism of the androgens, therapeutics and the preparation of the male sex hormone. There is a bibliography of 260 titles. Indexes, however, are lacking; they are essential in this type of survey monograph.

Electrophoresis in Physiology. By Lena A. Lewis, Ph.D., Research Division, Cleveland Clinic, Cleveland, Ohio. 8°, paper, 66 pp., with 5 illustrations. Springfield, Illinois: Charles C Thomas, 1950. $1.85. (Publication No. 89, American Lecture Series.)

This monograph constitutes a survey of the pertinent literature on the various uses of electrophoresis in physiology. There is a bibliography of 107 titles, and indexes of authors and subjects.

Trabajos del Sanatorio Antituberculoso de Ofra y del Dispensario Central de Santa Cruz de Tenerife, Canarias (Espana). Director: Dr. T. Cerviá. Volumen IX, Años 1948–1949. Santa Cruz de Tenerife: Imprenta Católica, 1950.

This report contains the statistics of the sanatorium for the years 1948 and 1949 and a number of papers on various aspects of tuberculosis. The series should be in all collections on tuberculosis.

Physical Examination in Health and Disease. By Rudolph H. Kampmeier, M.D., associate professor of medicine, Vanderbilt University School of Medicine, visiting physician to Vanderbilt University Hospital and chief of the Medical Outpatient Service, Vanderbilt University Hospital, Nashville, Tennessee. 8°, cloth, 821 pp., with 550 illustrations. Philadelphia: F. A. Davis Company, 1950. $8.00.

This textbook is written primarily for students and is based on the lectures given by the author at the Vanderbilt University School of Medicine. The text is arranged according to the organs and regions of the body. The preliminary chapters discuss the case history of the individual; there is a special chapter on the body action by Dr. William F. Orr. Terminology is emphasized throughout the text, with the object of increasing the vocabulary of the student. There is an extensive index, and the book is well published.

Principles of General Psychopathology: An interpretation of the theoretical foundations of psychopathological concepts. By Siegfried Fischer, M.D., clinical instructor in psychiatry, University of California. 8°, cloth, 327 pp., with illustrations. New York: Philosophical Library, Inc., 1950. $4.75.

This new book of psychopathology is divided into four parts. Part one, on the fundamentals of psychopathological concepts, in twelve chapters discusses the disturbances of perception, thought, memory, attention, apperception, orientation, emotion, drive and volition, intelligence, language, consciousness and unconsciousness, phantasy, and movements of expression and empathy. This part comprises 186 pages of the text.

Part Two deals with comprehensible and causal connections, and includes dynamic psychopathology, neuroses and hysteria. Part Three is on syndromes (symptom-reflexes); and Part Four discusses the abnormal personality in five chapters, including personality and psychoses, and the psychopathic and neurotic personality. There is a short bibliography and indexes of names and subjects. The book is well published, and should be in collections on the subject.

Bacterial Polysaccharides: Their chemical and immunological aspects. By Martin Burger. 8°, cloth, 272 pp. Springfield, Illinois: Charles C Thomas, 1950. $6.00.

This comprehensive monograph discusses the polysaccharides of the various species of bacteria. The pertinent literature is surveyed, and lists of references are appended to the various chapters. There are indexes of authors and subjects. The monograph should be in all collections on bacteriology and infectious diseases.

Quakers in Science and Industry, being an account of the Quaker contributions to science and industry during the 17th and 18th centuries. By Arthur Raistrick, M.Sc., Ph.D. 8°, cloth, 361 pp., with 16 illustrations and a frontispiece. New York: Philosophical Library, Inc., 1950. $6.00.

The section on the Quakers in science and medicine in this history should interest medical historians. The Quaker botanists, naturalists and physicians are dealt with in two chapters. The names of John Bartram, John Fothergill, John Coakley Lettsom, William Woodville and Benjamin Waterhouse should be familiar to all students of medical history. There is a good index, the portraits are excellent, and the book is well published. It is recommended for all medical-history collections.

NOTICES

TRAINING PROGRAM AT VA HOSPITAL

Several openings are available in the residency training programs in neurology and in psychiatry at the Veterans Administration Hospital, Coatesville, Pennsylvania. The programs, organized by the Philadelphia Deans Committee, have been approved by the American Medical Association. Both residencies cover a period of three years or less, depending on the previous experience of an applicant, and are designed to prepare residents for certification in neurology or psychiatry by the American Board of Psychiatry and Neurology. The programs include rotation through the Veterans Administration Hospital, Coatesville, Veterans Administration Mental Hygiene Clinic, Philadelphia, the Philadelphia General Hospital and university and hospital out-patient clinics associated with Philadelphia's five medical schools. Applications should be sent to Vincent T. Lathbury, M.D., Executive Psychiatrist, Philadelphia Deans Committee, 111 North 49th Street, Philadelphia 39, Pennsylvania.

ANNOUNCEMENTS

Dr. Salvatore V. Bonanno announces the opening of his office for the practice of surgery at 42 Lincoln Street, Framingham.

Dr. Jack S. Parker announces the opening of an office for the practice of general and pediatric surgery at 3 Seaward Road, Wellesley Hills, Massachusetts.

Dr. Herbert I. Posin announces the opening of an office for the practice of general psychiatry at 375 Commonwealth Avenue, Boston.

SPRINGFIELD ACADEMY OF MEDICINE

A meeting of the Springfield Academy of Medicine will be held in the Woman's Club Building, 43 Spring Street, on September 11 at 8:30 p.m. A symposium on maternal death will be presented. Dr. Benjamin Tenney, Jr., professor of obstetrics and gynecology, Boston University, will act as moderator.

(*Notices concluded on page xv*)